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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to phenol. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in September of 1980.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of phenols in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to phenols for various subpopulations.

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AN EXPOSURE AND RISK ASSESSMENT FOR PHENOL

by

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1.a

FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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Office of Water Regulations and Standards

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The materials balance for phenol (Chapter III) was adapted from a draft report by Versar, Inc., produced under Contract 68-01-3852 to the Monitoring and Data Support Division, Office of Water Regulations and Standards, U.S. EPA.

I. TECHNICAL SUMMARY

RISK ASSESSMENT

Environmental exposure of humans to concentrations of phenol at levels equivalent to those causing effects (as extrapolated from studies on laboratory animals) appears to be rare. However, one particular effects study reported chromosomal damage to mice at concentrations approximately four orders of magnitude lower than other effects levels. This particular study warrants further exploration due to the variance of its effects level from other reported effects levels. If the conclusions of the chromosomal study are correct, a large fraction of the human population may be exposed to phenol levels equivalent to the level which elicited chromosomal damage in mice. In addition, investigation of the similarities between humans and laboratory mice in regard to chromosomal damage and the significance of chromosomal damage in human populations is needed.

Comparison of phenol exposure levels for humans in most subpopulations with effects levels (excluding the chromosomal damage study) shows a "safety margin" of approximately one order of magnitude or greater. Exposure levels for other subpopulations (users of medicinal products, people exposed to multiple sources of phenol) are approximately five times below effects levels. The size of these other subpopulations is expected to be very small.

Phenol is rapidly metabolized and excreted from the body. This is an important factor in reducing possible harm from long-term exposure to low concentrations. There is no evidence at this time that phenol is carcinogenic when administered orally to mice or rats; however further testing has been recommended.

Aquatic organisms only rarely appear to be exposed to concentrations of phenol in surface water at levels equivalent to concentrations causing adverse effects in laboratory bioassays. Monitoring of environmental concentrations of phenol in surface water is limited, however; additional monitoring is needed to confirm this conclusion. Based on existing monitoring data, sublethal and lethal effects levels for fish and invertebrates are higher than exposure levels by approximately one and two orders of magnitude, respectively. Environmental fate data indicate that phenol is rapidly degraded by microbial populations which would reduce the likelihood of long-term persistence in surface water.

HUMAN EFFECTS AND EXPOSURE

Phenol is readily absorbed from all routes of entry, distributed throughout the body, metabolized and rapidly excreted from the body. The biological half-life of phenol in man is approximately 3.5 hours.

Acute lethal values for phenol range from approximately 200 mg/kg to 700 mg/kg, regardless of the route or species. The cat appears to be the most sensitive species (oral LDLo 80 mg/kg), probably as a result of significant metabolic differences in the manner phenol is detoxified in this species. Slight to moderate kidney damage and slight liver changes have been reported in rats given 135 daily doses of 100 mg/kg phenol by gavage. Similar treatment with 50 mg/kg produced slight damage after 135 doses but not after 20 doses. Rats, however, have been able to tolerate much larger doses in drinking water (56 mg/rat/day or ∿280 mg/kg for a 200-g rat), probably due to its rapid metabolism as well as the intermittent nature of dosing in contrast to exposure by gavage.

There are no indications that phenol is carcinogenic by the oral route. Skin application of phenol, however, is tumorigenic in sensitive strains of mice but not in standard inbred strains of mice. The tumorogenic activity of phenol appears to be associated with its irritancy and subsequent skin hyperplasia.

Lethal mutations in *Drosophila* (fruit fly) are induced by exposure to phenol. In addition a significant increase in the incidence of chromosomal defects in a dose-related manner in spermatogonia and spermatocytes was observed in mice given phenol by gavage at dosage levels as low as 6.4 ug/kg/day. Furthermore, the data from this particular study suggest incremental increases in the incidence of chromosomal aberrations occur in consecutively treated generations. However, the treatment schedule utilized and the lack of data reporting prevent assessment of the significance of this finding. No indications of teratogenicity have been found.

The lowest reported oral lethal dose of phenol in man is one gram, but the majority of lethal values are in the 5- to 40-gram range. Central nervous system disturbances together with peripheral vasodilation result from an acute lethal dose of phenol leading to sudden collapse and unconsciousness. Death is due to respiratory arrest. Ingestion of non-lethal amounts of phenol can result in burning in the mouth, mouth sores, headache, vomiting, diarrhea, back pain, paresthesia, and production of dark urine (probably from oxidation products of phenol). Recent reports have also linked phenol to the production of cardiac arrhythmias during chemical face peeling procedures used in clinical treatments.

Aside from the issue of its mutagenicity, the rapid clearance of phenol from the body, its relatively high lethal dose, and the fact that small amounts of phenol are produced endogenously indicate that man can handle levels normally present in U.S. drinking water with no untoward effects. Further work needs to be done to validate the single report of increased chromosomal aberrations in phenol-treated mice and in particular, to clarify the finding of increased numbers of aberrations in consecutively treated generations of mice.

Numerous uncertainties are involved in estimates of exposure to phenol because of a deficiency in monitoring data. The use of phenol-containing products, especially mouthwash and lozenges, appears to be the largest consumer exposure in terms of exposure level, although presumably on a short time scale. Ingestion of contaminated well water may result in an equivalent exposure level but the duration of this level is dependent on the persistence of phenol in the groundwater. Other water supplies appear to contribute to a very small exposure level through ingestion. Chemical laboratory workers comprise a subpopulation potentially exposed to levels equivalent to those from use of phenol-containing mouthwash, however, assumably laboratory exposure occurs over a long period of time. In both cases the subpopulations are expected to be small. Ingestion in food and dermal absorption from cosmetics may contribute to a more continual exposure for a larger subpopulation. Ingestion of fish or smoked meat and inhalation along highways may each represent an exposure of up to 6 mg/day, using worst case assumptions.

Separate exposure routes were combined into various scenarios to estimate total exposure. This assumes equal absorption efficiency for all routes and upper limit exposure levels. The scenarios quantified include a worst case (combination of all exposures), groundwater contamination, medicinal use, laboratory exposure and the general population. Associated daily exposure levels with these scenarios were, respectively, 520 mg, 247 mg, 354 mg, 82 mg and 7 mg of phenol.

AQUATIC EFFECTS AND EXPOSURE

The Ambient Water Quality Criteria document for phenol states that the available data for phenol indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 10.2 and 2.6 mg/l, respectively. Toxicity may occur at lower concentrations among species that are more sensitive than those tested.

The lowest concentration of phenol associated with toxic effects is 0.1 mg/l, in Daphnia magna. The lowest acute level was for a species of insect (Baetis), a 48-hour LC_{50} of <1.5 mg/l. The juvenile rainbow trout was the most sensitive fish tested; LC_{50} values were reported at 5.0 mg/l. The grass frog was the only non-piscine vertebrate tested; lethal toxicosis in embryos was reported at 0.5 mg/l phenol.

Other toxic effects in addition to mortality have also been observed such as decreased reproductive rate and fecundity in Daphnia, loss of balance in pike, lack of pigmentation in developing sturgeon prolarvae, delayed hatching in bream, and reduced feeding in clams. Not all effects are obviously detrimental, however; Daphnia pulex grew faster in a solution of 0.1 mg phenol/l than under control conditions and some species had greater hatching success in very low concentrations of phenol.

The toxicity of phenol to marine organisms has been only briefly studied. The effects levels reported are in the same approximate range as for freshwater species. No community or population studies (laboratory or field) are available for both salt and freshwater ecosystems. The toxicity of phenol to aquatic life is influenced by various environmental factors. Water temperature is the most extensively studied variable, yet laboratory results present variations between species as to its general effects. In most cases, the organism is more sensitive to phenol as temperature increases, although high and low temperature extremes appear to be detrimental. The anadromous rainbow trout perished at lower phenol concentrations as salinity increased, suggesting that salmonoid populations might be more sensitive when their migrations bring them into estuaries. Crucian carp were more sensitive to phenol at both pH extremes.

Phenol concentrations of significance to aquatic life are few and short-term, based on the limited available data. Monitoring data reported 72% unremarked (above detection limit) ambient levels in U.S. fresh surface water less than 0.01 mg/l. The Tennessee and Ohio River basins had the highest maximum levels. Few data were available for estuarine or marine waters.

Industrial and wastewater treatment effluents often have high levels of phenol. Some of the higher U.S. levels may be for phenolics and not specifically phenol; therefore, the actual phenol concentration would be lower than the reported values. According to the results of the EXAMS model, which simulates the continual discharge of phenol into selected "average" aquatic systems, typical effluent levels would result in water column concentrations lower than 50 ug/l, with the majority less than 10 ug/l. The results are dependent on certain assumptions, including a high microbial degradation rate, which may not be applicable to all situations. Therefore, the results are not meant to be predictive.

FATE PATHWAYS AND ENVIRONMENTAL DISTRIBUTION

The fate and distribution of phenol following environmental release depends on the form of emission, the receiving medium, and various environmental factors. The critical environmental pathways describing the behavior of phenol releases include discharges to surface water, emissions to air, transport from air to water/soil, discharges to POTW's, releases to soil, and chlorination during water treatment.

Surface Water: Approximately 30% of all known environmental releases of phenol are made to surface water, primarily by POTWs, petroleum refiners, phenolic resin and bisphenol A producers, and certain producers of phenol itself. The most significant fate process affecting phenol in surface water is biodegradation. A half-life of 3.5 days was reported under field conditions in a river. Laboratory studies confirm a rapid removal rate especially under acclimated conditions and at high temperatures. Numerous microfloral species have been identified as capable of degrading phenol. There is some evidence that phenol may undergo photolysis under environmental conditions. The processes of hydrolysis, oxidation, adsorption, and volatilization do not appear to be significant with respect to phenol concentrations in surface water.

Bioaccumulation of phenol has been studied in aquatic organisms. Absorption is the primary route of intake. Phenol concentrations of 14 to 156 ug/gram of body weight were reported for goldfish exposed to 10-100 mg/l phenol for 1-5 days. Bioconcentration factors for phenol were low, ranging from 1.2 to 2.3. Accumulation of phenol occurs primarily in the gall bladder, liver, and visceral organs. Higher vertebrates, such as mammals, detoxify phenol by forming conjugation products with glucuronides and sulfates; fish do not appear to possess this mechanism, rather bronchial diffusion and biliary excretion are the mechanisms of decreasing the phenol body burden of these organisms. Biomagnification of phenol does not appear to be significant because of its generally low degree of accumulation in tissue.

A monohydric phenols river spill from a benzene sulfonation plant was monitored and the results showed 93% of the initial 28 mg/l phenol concentration was reduced significantly in six days. Associated with the high rate of phenol degradation was a temporary deficit in oxygen levels which contributed to the toxic effects resulting from the spill. Phenol concentrations may show seasonal variation, higher during the winter than summer due to a decreased rate of microbial degradation at low temperatures. A river receiving continuous discharges from a petroleum refinery and a cumene peroxidation plant had summer phenol levels approximately one order of magnitude lower than winter levels; however, some of the variation could have been attributable to differences in the waste characteristics and discharge levels.

A continous discharge of 3 kg/hr of phenol into a eutrophic lake and turbid river (1 km length) was simulated by the EXAMS model. Equilibrium water column concentrations were 1 to 3 ug/l. The self-purification time for both systems was approximately 3 to 4 hours due to biodegradation in the eutrophic lake and physical transport out of the modeled reach in the river system. Sediment concentrations were approximately 10 ug/kg and 5×10^{-4} ug/kg (dry weight) in the river and lake systems, respectively.

Monitoring data in surface water are limited for phenol. The STORET data base reports a total of approximately 600 observations for thirteen major river basins between 1978 and 1980. Mean concentrations ranged from 0.004 to 660 ug/l with a maximum of 6,794 ug/l. Sediment levels (348 observations) averaged around 102 mg/kg (unremarked data) with a maximum value of 454 mg/kg. Concentrations as high as 3,000 mg/l have been reported in effluents; however, most levels were reported at less than 1.0 mg/l.

Emissions to Air: Approximately 64% of phenol releases are to air, predominately from combustion of wood and automobile exhaust. Phenol producers, consumers, and the transport and storage of phenol are responsible for other atmospheric releases. Most releases are presumably in vapor form or adsorbed onto particulate matter. Phenol is subject to rainout, photolysis and photooxidation and its estimated atmospheric lifetime is several days. In urban areas, phenol levels fluctuate diurnally with higher levels during the day. These levels were attributed to higher traffic volume and industrial activity at that time of the day. The highest reported phenol concentration in urban air was 289 ug/m^3 reported in Frankfurt, Germany. No comparable U.S. levels were reported. No data were available for atmospheric concentrations in rural areas.

Rainout: Atmospheric emissions of phenol not photodegraded are subject to rainout with transfer to land or surface water. Rainwater concentrations were estimated initially at 1 to 10 mg/l in the vicinity of a source; however the concentrations are expected to be reduced as rainfall continued. No monitoring data were available reporting phenol levels in rain. However, European industrial areas are reported to have higher rainfall concentrations than do rural areas but the levels were not quantified.

Fate in POTW's and Wastewater Treatment: Phenol in untreated and treated waste streams is discharged to POTW's by various industrial sources. Natural background levels of phenol also contribute some portion of the total loading. Phenol concentrations in POTW influents were reported at 0.001 to 0.2 mg/l. Common treatment processes are successful in removing phenol including activated sludge, trickling filters, and various chemical treatments. Biodegradation in sludge is very effective, especially in activated sludge at concentrations less than 10 mg/l. The decay rate is sometimes inhibited at levels exceeding this concentration. At approximately 500 mg/l an activated sludge system experienced a sharp disruption of microbial activity. The optimum pH range is 6 to 9.5 and adequate essential nutrient concentrations must be present.

A number of primarily tertiary treatment methods have been found to be effective in degrading phenol. These include treatment with chlorine, hydrogen peroxide, potassium permanganate, ozone, and iron ferrate. These methods are not expected to be very common at POTW's and variable in wastewater treatment facilities depending on the industry subcategory.

A high removal efficiency of greater than 90% was reported for phenol in four POTW plants in a field study evaluating POTW treatment processes. Efficiencies were not reported for the other three plants studied.

Soil to Groundwater and Surface Water: A small percentage (approximately 6%) of the environmental releases of phenol are known to associate with disposal of wastes on land. Major known sources include sludges resulting from the synthesis of phenol. Phenol has a relatively low affinity for adsorption onto soil and a high solubility. Therefore, some portion of the releases to land may reach either ground or surface waters unless significantly reduced a priori by biodegradation or other fate processes.

Biodegradation is expected to be the most important fate process determining phenol concentrations in biologically active soil. Phenol volatilization from soil does not appear to be significant. No specific information describing chemical oxidation nor complexation of phenol was available. Adsorption onto organic matter and clay is low.

Attenuation and persistence of phenol in soil in the vicinity of phenol sources were indicated by two field studies. In a peat soil, the influence of phenol was limited to within 500 m of a catchment pit which indicated, in contradiction of laboratory results, strong adsorption onto the highly organic peat. In the second study phenol spilled onto the soil surface reached groundwater supplies where it persisted for 19 months. The substratum in the vicinity of the spill was sand, gravel and undifferentiated dolomite. There was no indication of adsorption onto the organic fraction of the soil surface layer; however. soil concentrations were not reported to confirm this.

Chlorination of Phenol and Formation of Chlorophenols: Phenol is one of the most reactive aromatics during chlorination and synthesis of chlorophenols is commonly reported during treatment of wastewater and drinking water. Based on a laboratory experiment conducted under optimal conditions for chlorination, an upper limit of 1.6 mg of phenol per liter of water will become chlorinated at typical wastewater treatment chlorine concentrations. Lower phenol concentrations, however, are probably more typically subject to reaction with chlorine due to system variability and stereochemical factors. Any specific conclusions about or quantification of the chlorination of phenol during POTW treatment of wastewater cannot be supported by the limited and inconsistent field studies available.

MATERIALS BALANCE

In 1978, approximately 1,216,100 kkg of phenol were produced in the U.S. Of this amount, 639,200 kkg (53%) was consumed in the U.S., 103,800 kkg (8%) was exported, and the remaining 473,100 kkg (39%) was assumed to have been placed in stocks. Phenol is isolated from coal tar and petroleum streams (1%) and produced synthetically by cumene peroxidation (90%), benzene sulfonation, and toluene oxidation (together comprising 9%).

Use of phenol as an intermediate in the synthesis of various organic chemicals is responsible for 95% of its total consumption. Products include resins (44%), bisphenol (17%), caprolactam (15%), methylated phenol (4%), plasticizers, adipic acid, nonylphenol, alicylic acid, dodecylphenol, 2,4-dichlorophenoxyacetic acid, pentachlorophenol, other alkylphenols, and other chlorophenols. The remaining 5% of phenol is used as a solvent in petroleum refining, in medicinal products, or exported.

Production and consumption-related activities are associated with approximately 19,018 kkg of annual phenol releases (~1.5% of the total amount produced in 1978). Of the total amount released, approximately 10% derives from consumption of phenol products, 13% from production, 54% inadvertently from residential wood combustion and gasoline combustion, 21% from POTW discharges and 2% from transport and export activities.

Phenol releases are distributed among environmental media with 64% (12,121 kkg) to air, 30% (5,668 kkg) to water and POTW's, and 6% (1,229 kkg) to land. Air emissions are primarily attributable to residential wood burning (7,224 kkg), gasoline combustion (2,280 kkg) and vent releases during the cumene peroxidation production process (1,630 kkg). Discharges to water are primarily from POTW's (4,000 kkg), petroleum refiners (384 kkg), and bisphenol A producers (187 kkg) in the form of cooling water blowdown, condensate, and extracted liquids. Major sources to land are the producers of phenol which dispose of contaminated sludge from column bottoms and evaporation vessels.

An estimated 4,000 kkg of phenol (based on reported POTW effluent levels) is discharged to surface water from POTW's. Approximately 1,010 kkg (25%) of the total amount can be related to specific sources, primarily petroleum refiners, phenolic resin and bisphenol A producers. The remainder of the phenol in the discharge originates from other unquantified man-made sources, natural sources such as decaying organic matter, and possibly as intermediate breakdown products from POTW treatment of more complex organics.

Among the areas requiring further investigation are the POTW discharge estimate, identification of sources to POTW's, the significance of indirect and natural releases to the overall environmental balance of phenol and better understanding and quantification of the land disposal practices for phenol wastes.

II. INTRODUCTION

The Office of Water Regulations and Standards, Monitoring and Data Support Division of the Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goal of the tasks under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This report is intended to provide a brief, but comprehensive, summary of the manufacture, use, distribution, fate, effects, and potential exposure and risk in regard to phenol. In order to make effective use of this report and to understand the uncertainties and qualifications of the data presented herein, several problems must be identified.

Phenol is produced for use as an intermediate in the manufacture of other substances, primarily phenolic resins, bisphenol A, caprolactam, and many organic chemicals. It is used directly to a lesser degree as a disinfectant in assorted products and as an analytical agent. The production and use emissions are widespread throughout the United States and limited monitoring data suggest that phenol is ubiquitous in the environment at low concentrations.

Although the physical and chemical properties of phenol are well understood, environmental fate and monitoring data are few. It is difficult, therefore, to predict and confirm the persistence of phenol in the environment. In order to better understand phenol's environmental behavior, published case studies of spills were examined and a model simulating various aquatic systems was implemented to model phenol's fate under conditions of continued discharge.

An important source of phenol to waterways is inadvertant discharge from coal and petroleum-using facilities of the "natural" phenol present in the fuel. In addition there are natural background levels of phenol found in water. These sources should be accounted for as best as possible in a materials balance; however, their releases have not been quantified and any values provided are rough estimates.

Another problem associated with phenol is its propensity for chlorination and formation of potentially more toxic and persistent compounds. Chlorination occurs primarily during wastewater treatment and drinking water treatment. This exposure assessment does not consider chlorination products of phenol; however, in a separate assessment under the same program, the environmental fate of and exposure to 2-chlorophenol, 2,4-dichlorophenol, and 2,4,6-trichlorophenol are addressed.

This report is organized as follows:

- Section III contains information on the production, consumption, discharge, and disposal of phenol.
- Section IV describes the environmental fate of phenol in five pathways originating from the point of release. Monitoring data, field studies, and, for surface water, an environmental fate model, were analyzed to supplement laboratory studies.
- Section V presents reported effect levels in humans and laboratory animals and exposure pathways for humans.
- Section VI discusses reported effects levels and exposure pathways for aquatic organisms.
- Section VII discusses risk of exposure to phenol for the general population and selected subpopulations of humans and aquatic organisms.

SECTION III.

MATERIALS BALANCE

A. INTRODUCTION AND METHODOLOGY

In this section, a materials balance for phenol is developed presenting information on production, consumption, and, where available, disposal of phenol in order to identify pathways of entry to the natural environment. The section is largely based on a report prepared for the U.S. EPA in January 1980 (Versar 1980). It is supplemented with information from other current and past EPA reports, other available relevant literature, and personal communications with individuals active in portions of the industry, U. S. EPA staff, and industry experts at Arthur D. Little, Inc.

For each major source of pollutant release, the environmental compartments initially receiving and transporting the material (e.g., air, land, water, etc.) were studied in order to determine the nature, location, and quantity of phenol released to the environment. There are many uncertainties inherent in this analysis: not all current releases have been identified, past releases have not always been well documented, and future releases are difficult to predict in terms of type, quantity, and location. Nevertheless, sufficient information is available to indicate the nature, scale (temporal and geographical), and general distribution of the environmental release of phenol.

B. MATERIALS BALANCE

Phenol is an organic chemical which is produced and used domestically. A moderate amount is exported and a small quantity is imported annually. In 1978, approximately 1,216,000 kkg were produced and 639,200 kkg (53% of total production) were sold domestically (U. S. International Trade Commission 1978). Of the remainder, 103,800 kkg (8%) were exported and it is assumed that the difference of 473,100 kkg (39%) was placed in stocks (U. S. Department of Commerce 1978). Production and consumption patterns for 1978 are presented in Table 1. Data are reported for phenol production from coal tar, petroleum streams, and from synthetic routes (cumene peroxidation, benzene sulfonation, and toluene oxidation). Amounts of phenol consumed for the synthesis of various derivatives (e.g., phenolic resins, bisphenol A, caprolactam, methylated phenol, plasticizers, adipic acid, nonylphenol, salicylic acid, dodecylphenol, 2,4-dichlorophenoxyacetic acid, pentachlorophenol, other alkylphenols, other chlorophenols) are also presented. These uses account for about 95% of total phenol usage. Non-consumptive uses (solvents and exports) are also reported.

TABLE 1. U.S. PRODUCTION AND CONSUMPTION OF PHENOL
(kkg, Estimated 1978)

Source	Annual Production (kkg)	Annual Consumption (kkg)
Synthetic Phenol Production	1,216,100[1]	
by Cumene	(1,108,850)[2]	
by Toluene Oxidation	(107,250)[2]	
by Benzene Sulfonation		
Natural Phenol Production from Coal Tar and Petroleum Operations	83	
Imports	83[3]	
Exports		103,825[3]
Stocks		473,075[2]
Phenolic Resins		288,020[2]
Bisphenol A		105,380[2]
Caprolactam		92,980[2]
Methylated Phenol		*
Plasticizer (Excluding Adipic Acid)		18,595[2]
Adipic Acid		12,400[2]
Nonylphenol		12,400[2]
Salicylic Acid		9,300[2]
Dodecyphenol		6,200[2]
2,4-D (2,4-Dichlorophenoxyacetic Acid)		6,200[2]
Pentachlorophenol		3,100[2]
Other Alkylphenols		15,500[2]
Other Chlorophenols		9,300[2]
Petroleum Refining		6,200[2]
Other		53,708[2]
Total	1,216,266	1,216,183

* Production data were not readily available for 1978; however, it is assumed that methylated phenol consumption is included in "Other" category.

Source:
1. U.S. International Trade Commission 1978.
2. Arthur D. Little, Inc., estimates extrapolated from Versar 1980.
3. U.S. Department of Commerce 1978.

Table 2 presents estimated environmental releases based on 1978 consumption patterns. Approximately 19,018 kkg of phenol were introduced into the environment of the United States in 1978 from numerous sources including phenol-producing processes, processes in which phenol is used as a feedstock in the manufacture of other products (consumptive uses), processes in which phenol is used as solvent, storage of phenol by producers and users, phenol loading and transport operations, and emissions of phenol from miscellaneous sources: timber products processing, leather tanning, pulp and paper mills, textiles manufacturing and indirect sources, iron and steel production, steam electric units, residential wood-burning and hand-stoked coal furnaces, and Publicly Owned Treatment Works (POTW's).

Of approximately 19,018 kkg of phenol entering the total environment, about 2,500 kkg (13%) emanated from production processes, 1,850 kkg (10%) resulted from processes that used phenol as a feedstock, and about 10,270 kkg (54%) were byproducts from miscellaneous sources. Of particular note is that residential wood burning and gasoline combustion contributed approximately 9,500 kkg (81%) of the nationwide phenol air emissions. The remainder of the releases were from POTW's contributing approximately 4,000 kkg (21%) and releases during storage of 392 kkg (2%). The total identified emissions (19,018 kkg) were 1.5% of the total 1978 U.S. production. Figure 1 shows the total annual phenol flow in the United States based on 1978 data from all recognized sources from production through disposal in the environment. In future years, it appears that the amount of phenol released into the environment will be proportional to its annual production. As the reality of the nation's energy crisis is more keenly perceived, the popularity of wood-burning stoves is likely to increase dramatically causing a con-concomitant increase in phenol air emissions.

The estimated distribution of phenol releases to the environment in 1978, as shown in Table 2 and Figure 1, is summarized below:

Environmental Compartment	Phenol Releases (kkg)	Percent of Total Release (%)
Air	12,121	64%
Water	4,658	25%
Land	1,229	6%
POTW's	1,010	5%

It is apparent that air and water are the major recipients of the various phenol releases.

1. Production

Before 1914, coal tar and petroleum streams (natural phenol) were the only sources of phenol. Today, synthetic phenol production accounts for 99% of U.S. phenol production. About 91% is produced by cumene

TABLE 2. ENVIRONMENTAL RELEASES OF PHENOL
(Estimated 1978)

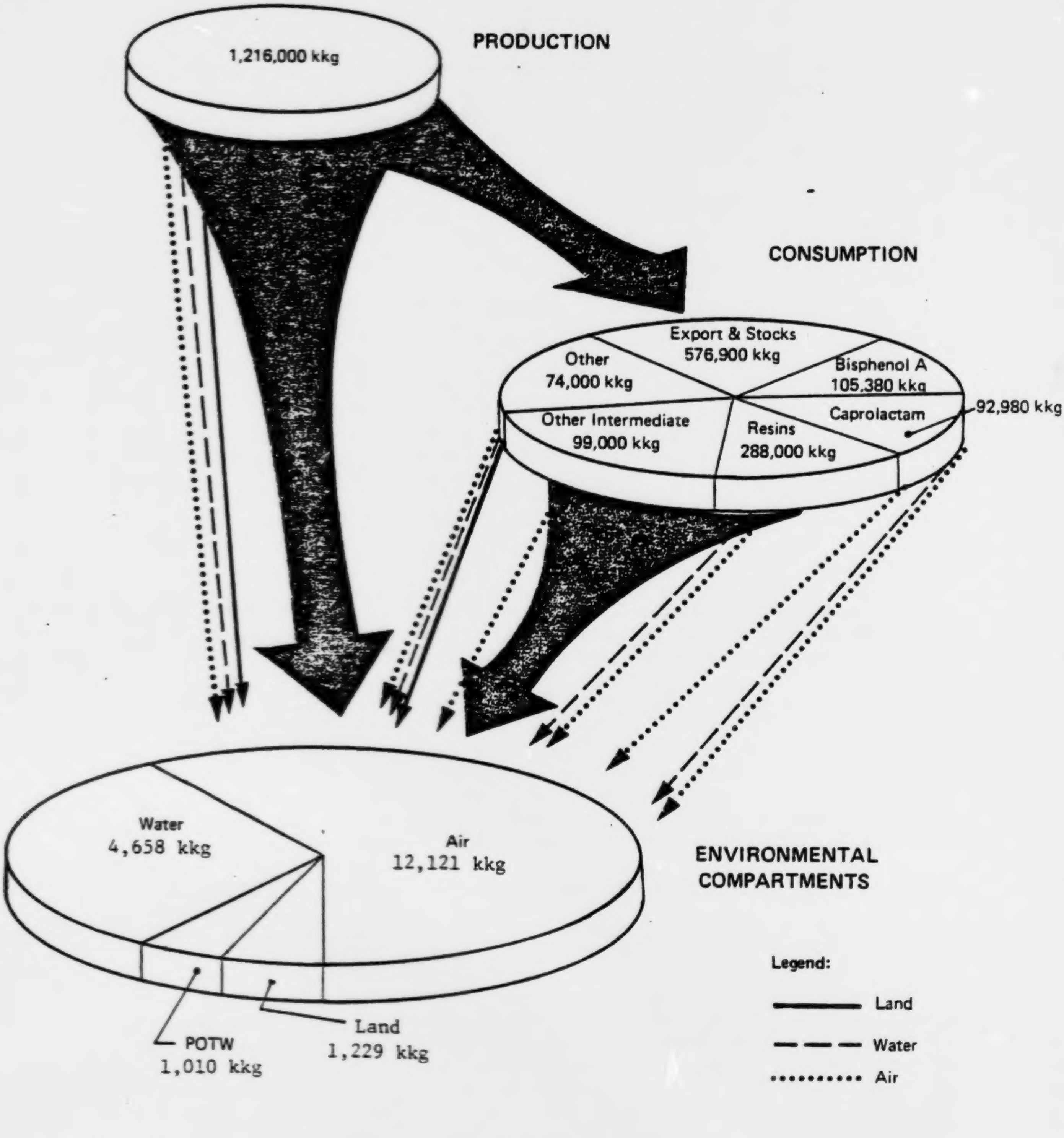
Source	Air	Direct Aquatic	POTW	Land
	Environmental Releases (kkg) to			
Production:				
by Cumene	1,630[9]	20[14]	18[14]	832[26]
by Toluene Oxidation			Δ	
by Benzene Sulfonation	Δ	Δ	Δ	397[27]
Transport	196[10]	Δ	-	-
Export	17			
Intermediate Consumption:				
Phenol Resins	77[10]	13[15]	572[15]	Δ
Bisphenol A	74[8]	187[16]	247[16]	Δ
Caprolactam	27[10]			Δ
Methylated Phenol	7[10]	Δ	Δ	Δ
Plasticizers (Excluding Adipic Acid)	5[10]	Δ	Δ	Δ
Adipic Acid	3[10]	Δ	-[25]	Δ
Nonylphenol	54[8]	Δ	Δ	Δ
Salicylic Acid	9[8]	Δ	Δ	Δ
Dodecylphenol	2[10]	Δ	Δ	Δ
2,4-D (2,4-Dichlorophenoxy-acetic acid)	2[10]	Δ	Δ	Δ
Pentachlorophenol	24[8]	Δ	Δ	Δ
Other Alkylphenols	4[10]	Δ	Δ	Δ
Other Chlorophenols	18[8]	Δ	Δ	Δ
Petroleum Refining	2[10]	384[17]	101[17]	
Other Use Categories	37[10]	Δ	Δ	Δ
Other Consumption:				
Timber Products	Δ	2[19]	2[19]	Δ
Leather Tanning	Δ	6[20]	64[20]	Δ
Textiles	Δ	1[21]	1[21]	Δ
Iron and Steel	Δ	3[22]	1[22]	Δ
Steam Electric	Δ	10[23]	-[23]	Δ
Residential Wood Burning	7,224[8]	-	-	-
Hand-Stoked Residential Coal Furnaces		-	-	-
Pulp and Paper Mills	-	32*	4*	
Automobile Exhaust	2,280			
POTW		4,000[24]	-	-
Storage	392[11]	Δ	Δ	Δ
Other	37			
Total	12,121	4,658	1,010	1,229

ΔNot currently available

Note: Numbers refer to derivations in Appendix A.

*U.S. EPA, Effluent Guidelines Data 1979, as yet unpublished.

Source: Arthur D. Little, Inc., estimates extrapolated from Versar 1980.


1,216,000 kkg
PRODUCTION
CONSUMPTION
Export & Stocks
576,900 kkg
Other
74,000 kkg
Bisphenol A
105,380 kkg
Caprolactam
92,980 kkg
Other Intermediate
99,000 kkg
Resins
288,000 kkg
Water
4,658 kkg
Air
12,121 kkg
ENVIRONMENTAL
COMPARTMENTS
POTW
1,010 kkg
Land
1,229 kkg
Legend:
Land
Water
Air

Note: Boundaries between receiving media are often undefined and/or changing; phenol apparently released to one medium may result in another.

Source: Tables 1 and 2.

FIGURE 1 MATERIALS BALANCE OF PHENOL

peroxidation, the remaining 9% by benzene sulfonation and toluene oxidation. The contribution by each process and the total production of phenol are shown in Table 3. Each process is described in detail in Appendix B. The names, locations, processes, and capacities of plants that produce phenol are presented in Table 4 and Figure 2.

The amount of phenol imported in 1978 was 83 kkg (U. S. Department of Commerce 1978).

2. Releases From Phenol Production

As previously mentioned, about 91% of phenol production is by the cumene peroxidation process. Known airborne emissions resulting from this process amount to between 3% and 17% of total airborne phenol emissions, that is, between 330 and 1,630 kkg annually in 1978 (see Table 2). Losses to water amount to 20 kkg, which is small compared with the total identified phenol aquatic discharges from all sources, about 4,600 kkg. An estimated 830 kkg were contained in solid waste. Known discharges to POTW's were estimated to equal 18 kkg.

Releases from phenol production by means of toluene oxidation and benzene sulfonation are treated collectively in this report; both processes together account for only about 9% of total phenol production. No releases to air, water, or POTW's have been identified, but a solid waste volume containing approximately 400 kkg was calculated (see Table 2). Only one company produces phenol by the benzene sulfonation process and only one plant uses the toluene oxidation process.

Only about 1% of phenol is recovered from the so-called natural sources of coal tar and petroleum operations. The quantity of airborne phenol emissions is unknown but possibly significant. The amount of phenol released to aquatic sinks is also unknown, but POTW discharges from this form of phenol production are considered negligible on a national scale, as is the volume of phenol-containing solid waste, due to the small total mass of phenol produced by this method.

3. Sources of Phenol Releases During Storage, Loading, and Transport

The estimated releases from storage, loading, and transport associated with production of phenol are 299 kkg (1978). This estimate is based on the supposition that 0.161 kg of phenol are lost per 1 kkg stored and transported (Delaney and Hughes 1979). No process-dependent discharges are included in this estimate. Phenol, bisphenol, and phenolic resins are stored and transported according to users' (consumers') needs in the form of liquids or solids. Frequently metal tanks are used if the material is stored or transferred in a particularly corrosive chemical form. Stainless steel tanks are utilized. Train or trunk tank cars transport the material depending on the quantity involved. Storage is maintained at both manufacturing points and the site of consumption. The primary potential for material release is during transfer activities in which the material passes between manufacturer and storage or transport

TABLE 3. TOTAL U.S. PRODUCTION OF PHENOL BY PROCESS, 1977 and 1978

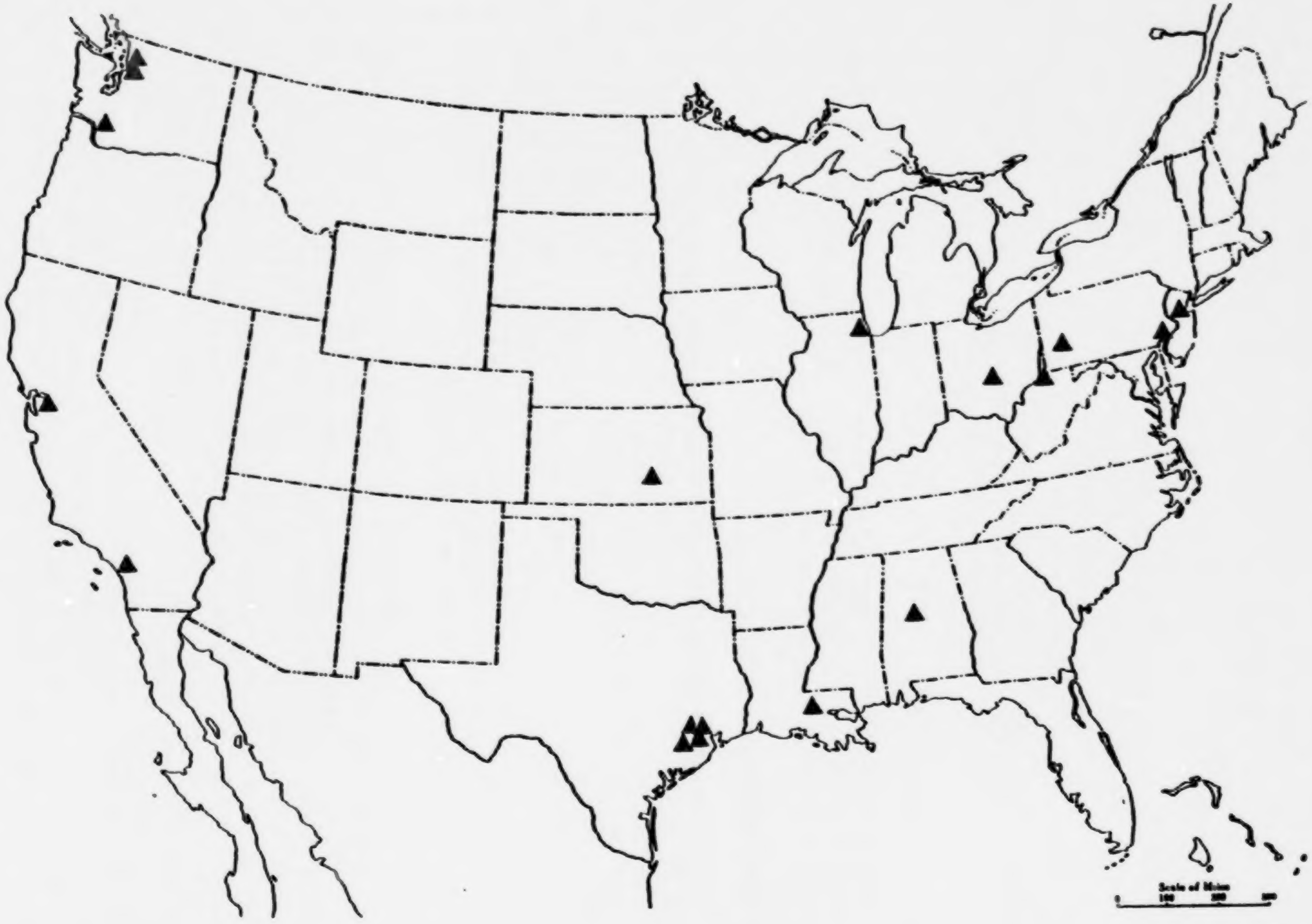
Process	1977 Phenol Production (kkg)	1978 Phenol Production (kkg)
Phenol from cumene peroxidation	966,709	1,108,850
Phenol from toulene oxidation	93,500	107,250
Phenol from benzene sulfonation		
Coal tar and petroleum operations	12,200	83
TOTAL	1,072,409	1,216,183

Source: Arthur D. Little Inc., estimates extrapolated from Versar (1980) and U.S. International Trade Commission (1978).

TABLE 4. U.S. PHENOL MANUFACTURERS

Company	Location	Process	1977 Annual Capacity (10^3 kkg)
Allied Chemical	Frankford, PA	cumene peroxidation	272
Clark Oil	Blue Island, IL	cumene peroxidation	40
Dow Chemical	Oyster Creek, TX	cumene peroxidation	211
Ferro Corp.	Santa Fe Springs, CA	coal tar and petroleum	-
Georgia-Pacific Corp.	Plaquemine, LA	cumene peroxidation	120
Getty Oil	El Dorado, KS	cumene peroxidation	43
Kalama	Kalama, WA	toluene oxidation	25
Koppers Co., Inc.	Follansbee, WV	coal tar	-
Merichem Co.	Houston, TX	petroleum	-
Monsanto Co.	Chocolate Bayou, TX	cumene peroxidation	227
Northwest Petrochemical Corp. (Stimson Lumber Co.)	Anacortes, WA	petroleum	-
Reichhold Chemical	Tuscaloosa, AL	benzene sulfonation	70
Shell Chem. Co.	Deer Park, TX	cumene peroxidation	227
Standard Oil Co.	Richmond, CA	cumene peroxidation	25
Stimson Lumber			
Union Carbide Corp.	Bound Brook, NJ	cumene peroxidation	68
	Penuelas, PR	cumene peroxidation	91
United States Steel Corp.	Clairton, PA	coal tar	147
	Haverhill, OH	cumene peroxidation	-
		Total	1,566 +

Source: Versar 1980 and U.S. International Trade Commission 1978.


Scale of Miles
0 100 200 300

▲ Penuelas, Puerto Rico

Sources: Versar (1980) and U.S. International Trade Commission (1978).

FIGURE 2 LOCATIONS OF U.S. PHENOL MANUFACTURERS

vessel or storage or transport vessel and user. Data are not available to accurately quantify this release.

Since roughly 0.006% of the total domestic phenol supply was imported in 1978, releases from unloading of imported phenol and transport to the point of consumption are assumed to be negligible.

4. Uses of Phenol

The utilization of phenol in the United States was 743,000 kkg in 1978 (see Table 1). This can be divided into consumptive and non-consumptive uses. Consumptive uses include processes in which phenol is chemically converted to another compound. Non-consumptive uses include exports and processes in which phenol is used as an end-product rather than as an intermediate. Table 5 shows the percentage of phenol used by each end use. Figure 3 presents a generalized pattern of phenol use.

a. Consumptive Uses

It is estimated that 633,000 kkg of phenol were consumptively used in 1978 (see Table 1). The major use was as a chemical intermediate in the synthesis of other organic chemicals.

i. Phenolic Resins

The manufacture of phenolic resins consumed 288,020 kkg (24%) of phenol in 1978 (see Table 1). Phenolic resins are the oldest synthetic polymers and are produced by reacting phenol, or substituted phenols, with an aldehyde. Almost all resins significant to industry are based on the reaction of phenol with formaldehyde. Two types of resins are produced: resols--a mixture of the two substances with an excess of formaldehyde--and novalaks--a mixture with a deficiency of formaldehyde. (Sittig 1975). Both types are manufactured by similar processes, described in greater detail in Appendix B. The locations of manufacturers of phenol resins are shown in Table B-1 and Figure 4.

The major end use for phenolic resins is as an adhesive in plywood. Thus, the demand for these resins is dependent on the housing industry. To a lesser extent, phenolic resins are used for the hardwood plywood market to produce waterproof bonds. Phenolic resins contribute to the weathering properties of low-cost, highly absorptive southern pine and enable it to be used instead of more expensive northern woods. Phenolic-bonded wood can be used indoors or outdoors, whereas wood bonded with protein or urea-formaldehyde adhesives cannot be used outdoors because the adhesives are not moisture resistant.

The second largest use for phenolic resins is in foundry resins (e.g., for use in casting automobile parts). These resins are also used in the comparison molding of plastic parts, laminating, thermal insulation, and protective coatings.

TABLE 5. U. S. PHENOL UTILIZATION[1]

Consumptive Uses	Percent of Total Phenol Utilization (%)
Phenolic Resins	39
Bisphenol A	14
Caprolactam	13
Methylated Phenol	4
Plasticizers	3
Adipic Acid	2
Nonylphenol	2
Salicylic Acid	1.5
Dodecylphenol	1
Other Alkylphenols	2.5
2,4-D	1
Pentachlorophenol	0.5
Other Chlorophenols	1.5
Non-Consumptive Uses	
Petroleum Refining (solvent application)	1
Exports	14
Total	100

---

[1] Based on annual utilization of 743,000 kkg.

Source: Table 1 in this report.

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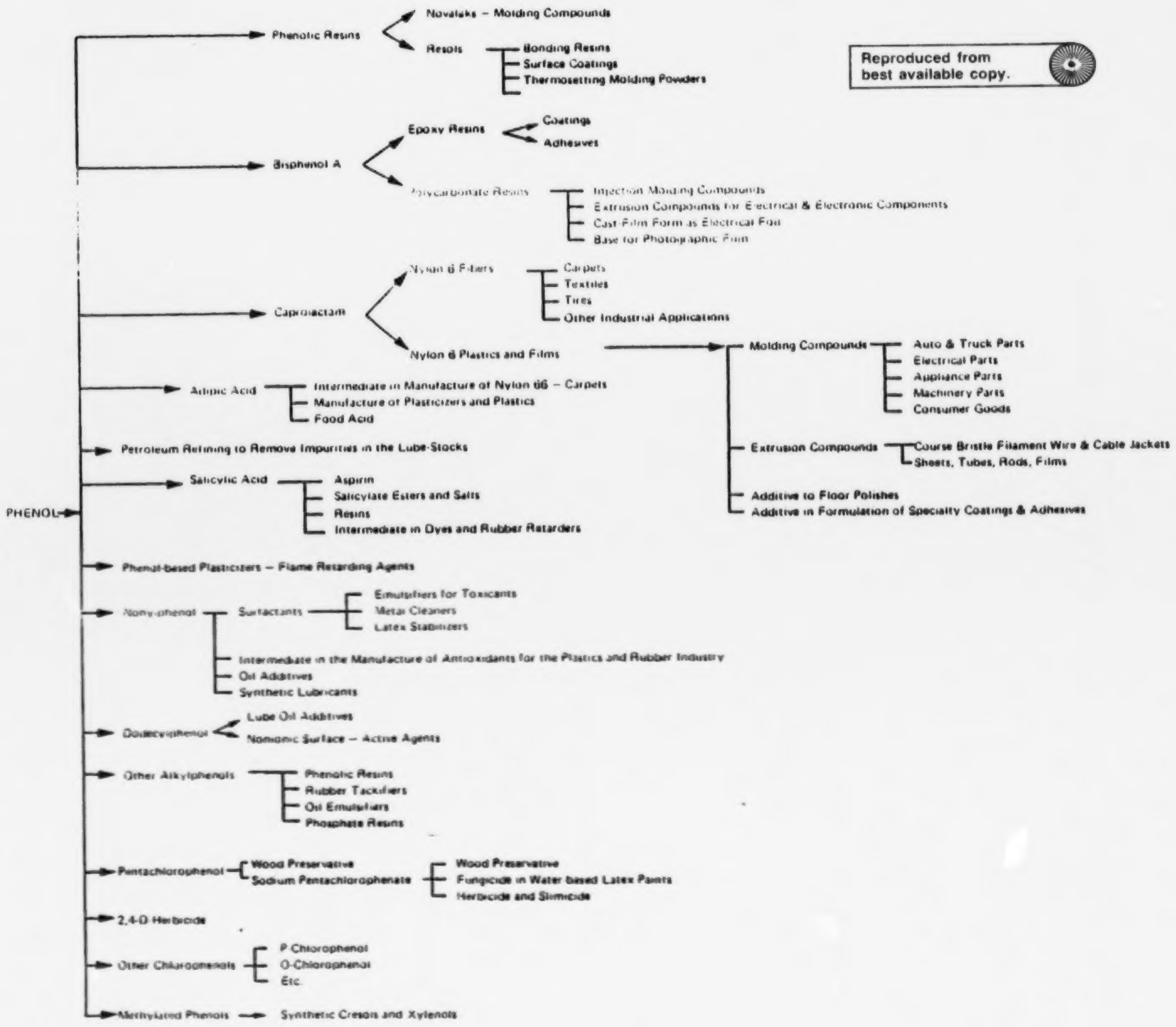
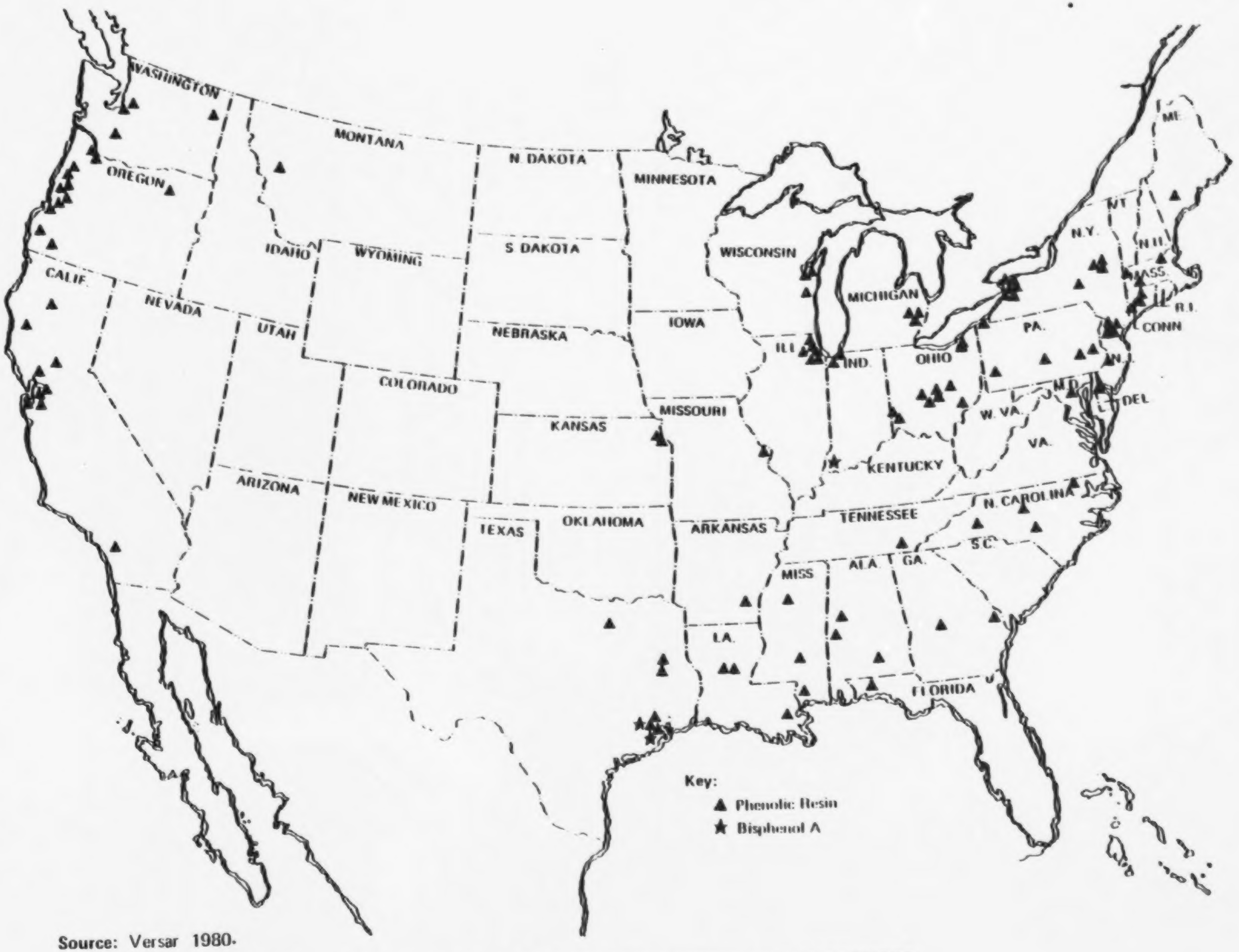

PHENOL
Phenolic Resins
Novalaks – Molding Compounds
Resols
Bonding Resins
Surface Coatings
Thermosetting Molding Powders
Bisphenol A
Epoxy Resins
Coatings
Adhesives
Polycarbonate Resins
Injection Molding Compounds
Extrusion Compounds for Electrical & Electronic Components
Cast Film Form as Electrical Foil
Base for Photographic Film
Caprolactam
Nylon 6 Fibers
Carpets
Textiles
Tires
Other Industrial Applications
Nylon 6 Plastics and Films
Molding Compounds
Auto & Truck Parts
Electrical Parts
Appliance Parts
Machinery Parts
Consumer Goods
Extrusion Compounds
Course Bristle Filament Wire & Cable Jackets
Sheets, Tubes, Rods, Films
Additive to Floor Polishes
Additive in Formulation of Specialty Coatings & Adhesives
Adipic Acid
Intermediate in Manufacture of Nylon 66 – Carpets
Manufacture of Plasticizers and Plastics
Food Acid
Petroleum Refining to Remove Impurities in the Lube-Stocks
Salicylic Acid
Aspirin
Salicylate Esters and Salts
Resins
Intermediate in Dyes and Rubber Retarders
Phenol-based Plasticizers – Flame Retarding Agents
Nonylphenol
Surfactants
Emulsifiers for Toxicants
Metal Cleaners
Latex Stabilizers
Intermediate in the Manufacture of Antioxidants for the Plastics and Rubber Industry
Oil Additives
Synthetic Lubricants
Dodecylphenol
Lube Oil Additives
Nonionic Surface – Active Agents
Other Alkylphenols
Phenolic Resins
Rubber Tackifiers
Oil Emulsifiers
Phosphate Resins
Pentachlorophenol
Wood Preservative
Sodium Pentachlorophenate
Wood Preservative
Fungicide in Water based Latex Paints
Herbicide and Slimicide
2,4-D Herbicide
Other Chlorophenols
P-Chlorophenol
O-Chlorophenol
Etc.
Methylated Phenols
Synthetic Cresols and Xylenols

Source: Versar 1980.

FIGURE 3 GENERALIZED FLOW PATTERN FOR PHENOL USE


WASHINGTON
MONTANA
N. DAKOTA
MINNESOTA
ME.
OREGON
VT
N.H.
N.Y.
IDAHO
WYOMING
S DAKOTA
WISCONSIN
MASS.
CALIF.
MICHIGAN
NEVADA
R.I.
IOWA
NEBRASKA
CONN
UTAH
PA.
ILL
OHIO
IND.
N.J.
COLORADO
MD
DEL
MISSOURI
KANSAS
W. VA.
VA.
KENTUCKY
ARIZONA
N. CAROLINA
NEW MEXICO
TENNESSEE
TEXAS
OKLAHOMA
ARKANSAS
S.C.
MISS
ALA.
GA.
LA.
FLORIDA
Key:
▲ Phenolic Resin
★ Bisphenol A

Source: Versar 1980.

FIGURE 4 LOCATION OF PHENOLIC RESIN AND BISPHENOL A PRODUCERS

Significant waterborne wastes emanating from the production of phenolic resins are water introduced with the raw materials, water formed as a product of the condensation reaction, caustic solution used for cleaning the reaction kettles, and blowdown from cooling towers (Sittig 1975).

ii. Bisphenol A

Bisphenol A is produced by the reaction of phenol with acetone. Production of bisphenol A consumed an estimated 105,380 kkg (9%) of the phenol manufactured in 1978 (see Table 1). Four companies produced bisphenol A in 1977. The names and locations of these plants are given in Table 6 and Figure 4.

A schematic flow diagram of the production of bisphenol A is shown in Figure 5. The phenol and acetone are added into the reaction vessel in a 3 to 1 molar ratio. Small amounts of catalyst promoter (methyl mercaptan) are added, and then the catalyst, dry hydrogen chloride gas, is bubbled through the mixture. The temperature is held at 50°C for 8 to 12 hours. A slurry of crystalline bisphenol A is produced.

A number of byproducts are formed in conjunction with the main reaction. In some plants these impurities are eliminated by batchwise crystallization. However, in at least one plant, continuous distillation and extraction crystallization are employed to purify the product. To produce purified bisphenol A, the slurry is transferred into a still where it is stripped of excess phenol and water. The overhead is decanted into an organic phase (consisting mainly of phenol which is recycled) and an aqueous phase. The latter is piped into the hydrogen chloride recovery unit, and contaminated water is sent to disposal (disposal practices unknown). Bottoms from the stripper are sent to a series of purification distillation chambers where excess phenol, isomers, and heavy ends are removed from the system for either recycle or disposal. Distillate from the last chamber is sent to the extraction operation, which produces a slurry of pure crystals. The filtrate from the centrifuge is partially recycled to the crystallizer, and the remainder is concentrated in an evaporator to produce liquid bisphenol A.

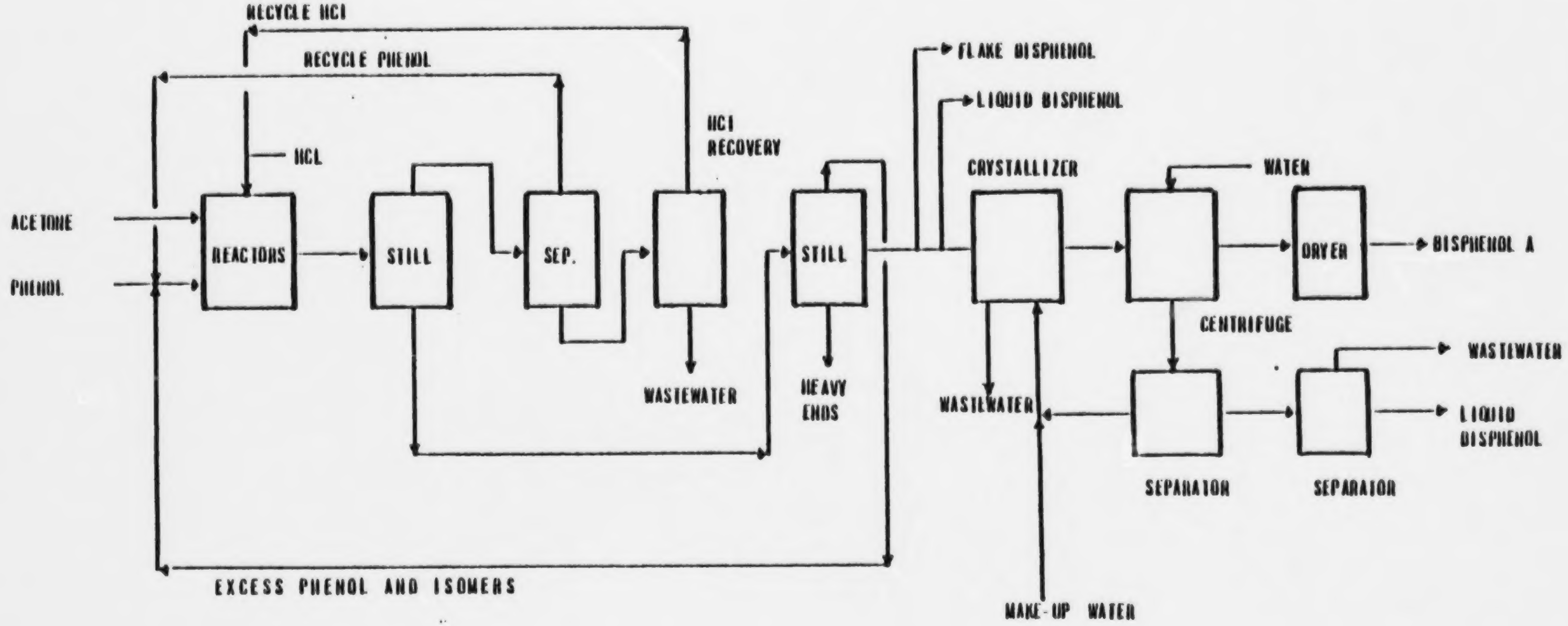
The major uses for bisphenol A are in epoxy resins and polycarbonate resins. The epoxy resins are mainly used for coatings and adhesives. Polycarbonate resins are used in injection molding, as an extrusion compound for electrical and electronic components, as electrical foil in the cast-film form, and as a basis for photographic film.

The major sources of waterborne wastes are the water separated from the hydrochloric acid recovery unit, the extracted aqueous phase from the crystallizer, and the condensate from the final evaporator. The organic wastes in this water are mainly phenol, bisphenol, and organic solvent. Organic vapor escaping from the final evaporator may contribute significant amounts of contaminants (U. S. EPA 1975).

TABLE 6. U. S. BISPHENOL A MANUFACTURERS

Company	Location	Annual Capacity (10^3 kkg)
Dow Chemical, U.S.A.	Freeport, TX	68
General Electric Company Plastics Business Division Engineering Plastics Product Dept.	Mount Vernon, IN	99
Shell Chemical Co.	Deer Park, TX	68
Union Carbide Corporation Union Carbide Caribe, Inc., subsid.	Penuelas, PR	32
Total		267

Source: Versar 1980.


RECYCLE HCl
RECYCLE PHENOL
HCL
ACETONE
PHENOL
REACTORS
STILL
SEP.
HCl RECOVERY
WASTEWATER
STILL
HEAVY ENDS
FLAKE BISPHENOL
LIQUID BISPHENOL
CRYSTALLIZER
WASTEWATER
WATER
CENTRIFUGE
DRYER
BISPHENOL A
WASTEWATER
LIQUID BISPHENOL
SEPARATOR
SEPARATOR
EXCESS PHENOL AND ISOMERS
MAKE-UP WATER

Source: U.S. EPA 1975.

FIGURE 5 BISPHENOL A PRODUCTION

iii. Caprolactam

The manufacture of caprolactam consumed 92,980 kkg (8%) of total phenol produced in 1978 (see Table 1). Allied Chemical Corporation in Hopewell, Virginia, is the only company in the United States to produce caprolactam from phenol; this plant has a capacity of 190,000 kkg of caprolactam. Two other companies that produce this chemical use cyclohexane instead of phenol as their starting material.

Cyclohexanone is the key intermediate in the caprolactam process. It is derived from phenol by catalyst hydrogenation and from cyclohexane by an air oxidation process (Lowenheim and Moran 1975). The catalytic hydrogenation of phenol produces both cyclohexanols and cyclohexanone. For cyclohexanol, a nickel catalyst is used; for cyclohexanone, palladium or carbon catalyst is employed. The caprolactam is produced in the Beckmann process by the addition of hydroxylamine sulfate to the cyclohexanone (Lowenheim and Moran 1975). Small amounts of phenol (estimated at 27 kkg annually) may be lost to the process water in the catalyst recovery unit, the wash tower, and the final product purification step (Lowenheim and Moran 1975).

Over 90% of the caprolactam produced is used to produce Nylon 6 fibers. The largest market for Nylon 6 fibers is carpets. Other uses are textiles and industrial applications such as tires. Caprolactam is also used to manufacture Nylon 6 plastics and films. These latter products are used to make molding compounds for automobile parts and appliance parts, extrusion compounds for coarse bristle filaments, and wire and cable jackets.

iv. Methylated Phenols

The production of methylated phenols consumed approximately 41,465 kkg of the phenol produced in 1977 (a 1978 number was not available). They are made by reacting phenol and methanol.

Methylated phenols are used in turn to produce synthetic cresols and xylenols. The xylenols are used as antioxidants for gasoline, lubricating oils, and elastomers. O-cresol is mainly used in the production of agricultural products and plastics and resins.

v. Plasticizers

Approximately 18,600 kkg (2%) of the phenol produced in 1978 were used to manufacture plasticizers (see Table 1). The most common phenol-based plasticizer is cresyl diphenyl phosphate. This plasticizer is used primarily to impart fire retardancy to polyvinyl chloride. Triphenyl phosphate, another phenol-based plasticizer, is used as a flame-retarding agent and as a plasticizer for cellulose acetate and nitrocellulose. Other phenol-derived plasticizers are octyl diphenyl phosphate, isodecyl diphenyl phosphate, and dibutyl phenyl phosphate.

vi. Adipic Acid

The manufacture of adipic acid consumed about 12,400 kkg (1%) of the phenol produced (see Table 1). Only Allied Chemical Corporation in Hopewell, Virginia, produces adipic acid using phenol as the raw material; this plant has a capacity of 13,600 kkg. The other five companies that produce adipic acid use cyclohexane as the raw material.

Adipic acid from phenol is produced by hydrogenation of phenol in the presence of nickel catalyst to cyclohexanol, which is then oxidized with nitric acid to adipic acid (Lowenheim and Moran 1975).

The major use of adipic acid is as an intermediate in the manufacture of Nylon 66. The carpet market offers the largest outlet for this nylon. Adipic acid is also used in the manufacture of plasticizers and certain plastics and as a food acid.

vii. Nonylphenol

About 12,400 kkg (1%) of the phenol consumed is used to manufacture nonylphenol (see Table 1). The names, locations, and capacities of the companies that produce nonylphenol are given in Table 7.

Nonylphenol is manufactured by liquid-phase alkylation of phenol with mixed isomeric nonenes in the presence of an acid catalyst. Premixed phenol and nonene are fed to an agitated, jacketed tank reactor where they react, in the presence of catalyst, at 50 to 100°C for a period of 30 to 120 minutes. In the reaction product, the parasubstituted derivative predominates, with smaller amounts of ortho and 2,4-dinonylphenols also present. Vaccum distillation is employed to separate the para-nonylphenol from the dinonylphenols (Lowenheim and Moran 1975).

Most of the nonylphenol produced is consumed in the manufacture of surfactants. Nonylphenol is converted into detergents either by ethoxylation or sulfonation. These detergents are used in specialty applications, such as emulsifiers for toxicants, metal cleaners, and latex stabilizers. Nonylphenol is also used in the plastics and rubber industry where it is an intermediate in the manufacture of antioxidants. The reaction of nonylphenol with formaldehyde produces compounds useful as oil additives and synthetic lubricants (Lowenheim and Moran 1975).

viii. Salicylic Acid

The manufacture of salicylic acid consumed slightly less than 1% (9,300 kkg) of the phenol produced (see Table 1). Four companies produce both technical- and medicinal-grade salicylic acid. One additional company produces only medicinal-grade salicylic acid. The names, locations, and capacities of these plants are given in Table 8.

TABLE 7. U.S. NONYLPHENOL MANUFACTURERS

Company	Location	Annual Capacity (10^3 kkg)
Borg-Warner Corp.	Morgantown, WV	27
Exxon Corp.	Bayway, NJ	13
Ferro Corp.	Sante Fe Springs, CA	0.9
GAF Corp.	Calvert City, KY	2
	Linden, NJ	9
Kalama	Kalama, WA	9
Monsanto Co.	Kearny, NJ	18
Rohm and Haas Co.	Philadelphia, PA	11
Rohm and Haas Texas, Inc., subsid.	Deer Park, TX	5
Schenectady Chems., Inc.	Oyster Creek, TX	23
	Rotterdam Junction, NY	10
Texaco Inc.	Port Neches, TX	16
Uniroyal Chem.	Naugatuck, CT	5
Total		149

Note: Some capacities also include that for other alkylphenols.

Source: Versar 1980.

TABLE 8. U.S. MANUFACTURERS OF TECHNICAL- AND MEDICINAL-GRADE SALICYLIC ACID

Company	Location	Annual Capacity (10^3 kkg)
Atomergic Chemetals Corp.[1] MWM Chems. Corp., subsid.	Plainview, NY	NA
Dow Chem. U.S.A.	Midland, MI	8
Monsanto Co. Monsanto Chem. Intermediates Co.	St. Louis, MO	9
Sterling Drug Inc. The Hilton-Davis Chem. Co. Div.	Cincinnati, OH	3
Tenneco Inc. Tenneco Chems., Inc.	Garfield, NJ	5
Total		25

[1] Produces only medicinal-grade salicylic acid.

Source: Versar 1980.

Salicylic acid is produced by reacting dry, powdered sodium phenate (made from phenol and sodium hydroxide) with excess carbon dioxide to produce sodium salicylate solution. This is next acidified with either hydrochloric acid or sulfuric acid to obtain salicylic acid (Morrison and Boyd 1970).

Salicylic acid is used primarily in the production of aspirin. It is also used in the production of salicylate esters and salts, in resins, as a dyestuff intermediate, and as a prevulcanization inhibitor.

ix. Dodecylphenol

Dodecylphenol is produced from phenol and propylene tetramer. It consists mainly of a mixture of p-alkylphenols derived from various isomeric branched-chain dodecylenes. The manufacture of dodecylphenol consumed about 6,200 kkg (less than 1%) of the phenol produced (see Table 1). Dodecylphenol is produced by four companies. The names and locations of the plants are listed in Table 9.

Approximately 95% of the dodecylphenol produced is used in the production of lube oil additives. The remainder is used in the manufacture of nonionic-surface active agents.

x. 2,4-D

The manufacture of 2,4-D (2,4-dichlorophenoxyacetic acid) consumed about 6,200 kkg (less than 1%) of the phenol produced in 1978 (see Table 1). There are nine companies that produce 2,4-D and its esters and salts. The names and locations of the plants are listed in Table 10.

The herbicide 2,4-D is manufactured by the reaction of sodium salt of 2,4-dichlorophenol (made by phenol chlorination) with monochloro acetic acid. 2,4-D is used as a weed killer and a defoliant. It is used extensively in the weeding of cereal crops, corn, sorghum, milo, sugar cane, coffee, pastures, range land, lawns, and unwanted growth.

xi. Pentachlorophenol

Pentachlorophenol production consumed about 3,100 kkg (less than 0.5%) of the phenol produced (see Table 1). Three companies produce pentachlorophenol. The names, locations, and capacities of the plants are listed in Table 11.

Pentachlorophenol is produced by chlorination of phenol or polychlorophenols in a batch operation. Phenol and chlorine are fed to a reactor where the chlorination is carried out in the absence of a catalyst until trichlorophenol is formed. At that point a metallic chloride catalyst (e.g., $FeCl_3$ or $AlCl_3$) is added to complete the reaction.

TABLE 9. U.S. DODECYLPHENOL MANUFACTURERS

Company	Location
Borg-Warner Corp. Borg-Warner Chems. Div.	Morgantown, WV
GAF Corp. Chem. Products	Calvert City, KY
Monsanto Co. Monsanto Indust. Chems. Co.	Kearny, NJ
Schenectady Chems., Inc.	Oyster Creek, TX Rotterdam Junction, NY

Source: Versar 1980.

TABLE 10. U.S. 2,4-D(2,4-DICHLOROPHENOXYACETIC ACID) MANUFACTURERS

Company	Location
Dow Chem. U.S.A.	Midland, MI
Fallek-Lankro Corp.	Tuscaloosa,AL
Imperial, Inc.	Shenandoah, IA
North American Phillips Corp. Thompson-Hayward Chem. Co., subsid.	Kansas City, KS
PBI-Gordon Corp.	Kansas City, KS
Rhodia Inc. Agricultural Div.	Portland, OR St. Joseph, MO
Riverdale Chem. Co.	Chicago Heights, IL
Union Carbide Corp. Agricultural Products Div. Amchem Products, Inc., subsid.	Ambler, PA Fremont, CA
Vertac, Inc. Transvaal, Inc., subsid.	St. Joseph, MO

Source: Versar 1980.

TABLE 11. U.S. PENTACHLOROPHENOL MANUFACTURERS

Company	Location	1978 Capacity (kkg)
Dow Chemical, U.S.A.	Midland, MI	12,000
Reichhold Chemicals, Inc.	Tacoma, WA	8,500
Vuleau Materials Company Chemicals Division	Wichita, KS	8,500
Total		29,000

Source: U.S. International Trade Commission 1978.

The majority of the pentachlorophenol produced is used as a wood preservative. A small amount is used to produce sodium pentachlorophenate, which is used as a wood preservative, as a fungicide in water-based latex paints, and as a herbicide (U. S. EPA 1975). A more detailed consideration of PCP can be found in EPA's Exposure Assessment on Pentachlorophenol (Scow, et al. 1980).

xii. Other Alkylphenols

Approximately 15,500 kkg (1%) of the phenol produced is used to manufacture alkylphenols other than nonylphenol and dodecylphenol (see Table 1). One of the most important of these chemicals is para-tert-butylphenol which is used in the preparation of phenolic resins, in rubber tackifiers, and in oil demulsifiers. Other important alkylphenols are the isopropylphenols, which are used in the production of phosphate esters, which in turn are used in the production of functional fluids and plasticizers.

xiii. Other Chlorophenols

Approximately 9,300 (less than 1%) of the phenol produced in 1978 was used in the production of chlorophenols other than 2,4-D and pentachlorophenol (see Table 1). Included among these chemicals are para-chlorophenol which is mainly used as an intermediate to produce other materials and ortho-chlorophenol which is recovered as a byproduct of para-chlorophenol. Para-chlorophenol is usually chlorinated further to produce higher chlorophenols.

b. Non-Consumptive Uses

Non-consumptive use is a minor category of phenol use. This category totaled 110,000 kkg in 1978, less than 15% of the total for consumptive use.

i. Petroleum Refining (Use of Phenol as a Solvent)

Approximately 0.5% (6,200 kkg) of the phenol produced in 1978 was used in petroleum refining (see Table 1). Phenol is produced at the refinery through catalytic cracking, crude distillation, and product finishing operations. In turn, phenol is used in solvent dewaxing operations where waxes are removed from lubricating oil stocks by promoting crystallization of the wax (U. S. EPA 1979d).

ii. Export of Phenol

Data provided by the Directory of Chemical Producers indicate 103,800 kkg phenol were exported in 1978.

c. User Storage, Loading, and Transport

Phenol has a relatively low volatility compared with many liquid hydrocarbons, but losses do occur during storage, handling, and transport because of volatilization, leakage, and spillage. The estimated amount of phenol released to the air because of user handling practices (390 kkg) is probably too low. Airborne emissions given in Table 2 for the various use categories probably overlap to some extent with emissions from handling procedures, but the amount of emissions reported from handling is sufficiently conservative that even with overlap it is unlikely that any inaccuracy would be significant.

It is known that used phenol drums are recycled into commerce and that some portion of these drums is available for purchase by the general public. Drum sellers and recyclers are supposed to flush phenol drums with sodium hydroxide in order to neutralize the phenol, and the washings are supposed to be collected in drums and sent to disposal. The flushing procedure is not always followed, however, and metallic drums containing up to about 250 g of phenol each are being purchased and used for various purposes by the general public (J. Warring, James T. Warring and Sons Barrel Company, personal communication, 1979). The amount of phenol lost to the environment each year is a function of the number of barrels discarded by phenol handlers and the degree to which drum recyclers adhere to the proper drum flushing practices.

5. Releases From Phenol Utilization

The single largest aquatic release due to phenol use is the discharge of about 187 kkg annually associated with the production of bisphenol A. In fact this is the single largest phenol release to water from production, use, and miscellaneous emissions, with the exception of POTW and petroleum refining discharges.

The airborne emission data for the various use categories are displayed in Table 2. The atmospheric emission has been derived on the basis of emission factors associated with generalized phenol-handling practices, and thus the emission number is proportional to the amount of phenol used in each use category. No equivalent data were available for those use categories for which only one airborne emission number is given in Table 2. Therefore, total airborne emissions due to phenol use (both in the production and actual consumer use categories) should actually be larger, possibly several times larger than shown in Table 2.

The specification of an airborne loss resulting from user storage, loading, and transport is to some extent redundant in that it repeats the numbers given in Table 2 as airborne emissions in the specific use categories. However, the emission factor for losses due to handling has actually been derived on the basis of producer handling losses. Since there are many more users than producers, and, consequently, since the number of handling and transfer operations must be much greater for users than for producers, the reiteration of any airborne emission from user handling practices seems warranted. In fact, the amount shown in Table 2 (392 kkg) is actually double the amount calculated on the basis of the producer-handling emission factor. On the basis of engineering knowledge, handling losses by users are deemed likely to be several times greater than 392 kkg, but there is no basis for using a higher multiplying factor than two in estimating an emissions amount. In fact, for the whole emission category of airborne emissions, the total emissions from the phenol use categories are certainly on the low side since the majority of airborne emissions listed are based only on handling losses and not on process or dissipative losses.

With regard to aquatic discharges from the use categories, few data were available. A significant amount of phenol is considered to be lost to water as a result of bisphenol A production; the amount lost (to both surface water and POTW's), is under 1% of the amount of phenol used in bisphenol A production. Also, approximately 585 kkg of phenol are lost to water, primarily to POTW's during the production of phenolic resins. The use of phenol as solvent in petroleum refining is associated with an effluent discharge of 485 kkg, of which 101 kkg goes to POTW's (U.S. EPA 1979d) (see also Appendix A No. 17).

Emissions due to export are attributed to transportation plus dockside loading. The air emission factor for these operations was based on the supposition that 0.161 kg of phenol are lost per kkg of material export (U.S. EPA 1979b). On this basis, the total estimated annual emissions to air are 6 kkg. No releases to water or land from phenol export have been identified and it is believed that any would be negligible.

The major discharge to POTW's identified is associated with petroleum refining, as is mentioned above. POTW emissions from the production of caprolactam and adipic acid have been identified as zero. Discharges to POTW's by other industries such as nonylphenol and chlorophenol producers are unknown.

No solid wastes from phenol uses have been identified, but many releases to land no doubt exist.

6. Miscellaneous Releases

The category "miscellaneous releases" covers those phenol emissions that emanate from a wide variety of sources where phenol use or production is entirely incidental. The sources can be divided into four categories: industrial sources, fuel combustion sources, POTW's, and medicinal products containing phenol.

a. Industrial Sources

Information on emissions of phenol from industrial sources is scanty and primarily deals with phenol in industrial effluents that are directly discharged to the environment or to POTW's. For the industrial categories of timber products processing, leather tanning, textile manufacturing, and iron and steel production, 12 kkg of phenol were directly discharged to the environment while 68 kkg were discharged to POTW's (see Appendix A No. 19-22). In all, the phenol contained in miscellaneous industrial effluents accounts for approximately 1% of all the phenol discharged to the aquatic environment.

b. Fuel Combustion Sources

Data on phenol emissions from fuel combustion are also sparse. Of note is the fact that emissions from residential wood burning were estimated to make up 60% of phenol air emissions nationwide (Versar 1980). There is some uncertainty in this estimate due to the lack of background data presented to support it. The type of combustion equipment, for example, or the kind of wood burned will influence the phenol concentrations emitted from a volume of wood. However, there is some evidence that a high value may be expected from domestic wood combustion. Phenol is an incomplete pyrolysis product from organic material (e.g., liquid fuels, plant material) and its generation is increased under the conditions of low temperature and oxygen typical of home wood-burning units (J. Allen, Battelle Columbus Laboratories, personal communication, 1980). More efficient, quick-burning processes (e.g., oil burners, wood-fired boilers used industrially) would have much lower associated phenol concentrations. Due to the increasing use of wood as a home-heating fuel, several groups are currently investigating wood-combustion emissions (e.g., Argonne National Laboratories, Monsanto, Battelle) (J. Allen, Battelle Columbus Laboratories, personal communication, 1980). Steam-electric units discharged 10 kkg directly to the aquatic environment from the ash handling subcategory (Appendix A No. 23).

Phenol is also a combustion product of liquid organic fuels, such as gasoline and diesel. Phenol emissions from combustion is variable depending on fuel variables: grade, source, refining technique, and seasonal variations in composition (PEDCo. 1977). The compound comes from the breaking of linkages connecting phenol groups, which are constituents of petroleum, and thus relasing them. Approximately 6 mg phenol were estimated to be released from each kg of exhausted diesel fuel and slightly greater than 6 mg per kg of gasoline fuel (estimated by Arthur D. Little, Inc., from information in Barber et al. 1964 and Hare and Bradow 1979). This would result in an air emission value on the same order of magnitude of that resulting from wood combustion, approximately 2,280 kkg/year. This estimate assumes an average engine efficiency and fuel composition on a national scale equivalent to those in the studies on which the estimates are based. It is based on a U.S. annual gasoline consumption value of 3.8×10^8 kkg (U.S. DOE 1979).

Emissions of phenol to air are also expected from combustion of other petroleum-based fuels such as aviation fuels, gas turbine fuel oils, kerosene, and others. It was not possible to estimate what these values are in this level of effort.

c. POTW's

An estimated 4,000 kkg of phenol were discharged directly from POTW's to the environment (see Appendix A No. 24) based on phenol levels in POTW effluents and the total U.S. POTW flow rate.

d. Medicinal Products Containing Phenol

A number of medicinal products, primarily skin lotions and sore throat remedies, contain phenol at levels of 0.5% to 4.75%. Table 12 lists the names of selected phenol-containing products; however, other products also exist and this tabulation is not inclusive. There was no information regarding the total amount of phenol consumed annually in production of these products.

C. AREAS FOR FURTHER RESEARCH

As discussed earlier in this section, this report is based on data from government documents (particularly publications issued by U.S. EPA), journals, and standard references and texts. In some cases the data are not associated with a high degree of confidence due to the small sample sizes supporting them or because they are outdated. Two areas where future investigations should be focused warrant mention.

Perhaps the weakest portion of the published literature is that dealing with phenol as an influent or effluent to POTW's. Up until now, it has only been possible to identify the sources of 1,006 kkg of phenol influents to POTW's, while an estimated 4,000 kkg of phenol are discharged into the aquatic environment by POTW's. It is expected that ongoing work under the aegis of U.S. EPA's Office of Water Regulations and Standards (through both its Effluent Guidelines Division and its Monitoring and Data Support Division) will identify fully the sources and amounts of phenol influents to POTW's, as well the sources and amounts of phenols discharged directly to the environment.

Another area that needs attention is the disposal of phenol as a solid waste in landfills. This phenomenon is likely to occur when phenol is adsorbed on a catalyst disposed of with the spent catalyst or (since phenol is a highly refractory organic compound) when phenol is disposed of as part of the sludge of industrial and/or wastewater pre-treatment and treatment plants. It is anticipated that the Background Documents (being prepared for the Office of Solid Waste both within and without U.S. EPA) on Processes Generating Hazardous Wastes, as defined in §250.14(b)(2) of the EPA Proposed Hazardous Waste Regulations under the Resource Conservation and Recovery Act (43 FR 58946, December 18, 1978), will be very useful as data sources for determining the fate of phenol in landfills.

TABLE 12. MEDICINAL PRODUCTS CONTAINING PHENOL

Product	% Phenol	Manufacturers
Campho Phenique®	4.75	® Glenbrook
Calamine Lotion®	1.0	® Mallinckrodt Inc., Penich & Co. (and others)
P & S Ointment/liquid®	1.0	® Baker Laboratories
Panscol Ointment®	1.0	® Baker Laboratories
Benadex Ointment®	1.0	® Fuller
Kip for Burns Ointment®	0.5	® Young's
Noxema Medicated Cream®	0.5	® Noxell
Tanurol Ointment®	0.75	® O'Neal, Jones and Feldman, Inc.
Dri Toxen cream®	1.0	® C. J. Walker, Co.
Peterson's Ointment®	2.5	® Peterson's Ointment Co.
Cepastat Mouthwash and Lozenges®	1.45	® Merrell-National Laboratories
Chloraseptic®	1.45	® Eaton Laboratories
Chloraseptic lozenges®	32.5 mg total phenol/ lozenge	® Eaton Laboratories

Source: U.S. EPA 1979c.

D. SUMMARY

In 1978, approximately 1,216,100 kkg of phenol were produced in the U.S. Of the total, 639,200 kkg (53%) was consumed in the U.S., 103,800 kkg (8%) was exported, and 473,100 kkg (39%) presumably was placed in stocks. Phenol is produced from coal tar and petroluem streams (1%) and from the following synthetic routes: cumene peroxidation (90%), benzene sulfonation, and toluene oxidation (together comprising 9%).

The primary use of phenol (95% of total consumption) is as an intermediate in the synthesis of various organic chemicals including phenol resins (44%), bisphenol A (17%), caprolactam (15%), methylated phenol (4%), plasticizers, adipic acid, nonylphenol, salicylic acid, dodecylphenol, 2,4-dichlorophenoxyacetic acid, pentachlorophenol, other alkylphenols, and other chlorophenols. The remaining 5% of phenol is used as a solvent in petroleum refining, in medicinal products, or exported.

Approximately 19,018 kkg of phenol (∿1.5 of the total amount produced in 1978) are released to the environment annually by production and consumption-related activities. Of the total amount released, approximately 10% derives from consumption of phenol products, 13% from production, 54% inadvertently from residential wood combustion and gasoline combustion, 21% from POTW discharges, and 2% from transport and export activities.

The estimated environmental distribution of phenol releases is 64% (12,121 kkg) to air, 30% (5,668 kkg) to water and POTW's, and 6% (1,229 kkg) to land. The most significant discharges to air result from residential wood burning (7,224 kkg) releasing natural phenols. Aquatic discharges are primarily from petroleum refiners (384 kkg), and bisphenol A producers (187 kkg) in the form of cooling water blowdown, condensate, and extracted liquids. Major contributors of phenol to land are the producers of phenol through disposal of contaminated sludge from column bottoms and evaporation vessels.

An estimated 4,000 kkg of phenol is discharged to surface water from POTW's based on reported POTW effluent levels. Only 1,010 kkg (∿25%) of the total amount can be accounted for, primarily from petroleum refiners. The remainder of the amount discharged originates from other unquantified intentional sources, natural sources such as decaying organic matter, and possibly as breakdown products from POTW treatment of more complex organics.

Further investigation is needed regarding the accuracy of the POTW estimate, identification of sources to POTW's, the significance of indirect and natural releases to the overall environmental balance of phenol, and land disposal practices of phenol wastes.

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SECTION IV.

FATE PATHWAYS AND ENVIRONMENTAL DISTRIBUTION OF PHENOL

A. INTRODUCTION

The following section describes the fate pathways of phenol in the environment following its intentional discharge or accidental release to water, air, and soil. Due to the short half-life of phenol under most environmental conditions, emphasis has been placed on its transformation and, because of its high reactivity and the potential for conversion to more harmful compounds, on its reaction products. Intermedia transfers are discussed in situations where they appeared to occur at a faster rate than transformation. Due to the limitations of the monitoring data base which describes phenol concentrations in environmental media, each major pathway description is followed by a short summary of reported measured concentrations in the medium considered in each pathway.

The section is organized into the following categories:

Pathway #1	Discharges to Surface Water
Pathway #2	Emissions to Air
Pathway #3	Air to Water/Soil: Rainout
Pathway #4	Fate in POTW's and Wastewater Treatment
Pathway #5	Soil to Groundwater and Surface Water: Leaching, Runoff
Pathway #6	Chlorination of Phenol and Formation of Chlorophenol

B. PHYSICAL PROPERTIES OF PHENOL

Table 13 summarizes some of the basic physical properties of phenol.

C. PATHWAY #1. DISCHARGES TO SURFACE WATER

1. Introduction

Phenol discharges to surface water are responsible for 25% of all phenol releases to the environment (see Section III). The major known direct dischargers are petroleum refiners (380 kkg), bisphenol A producers (approximately 187 kkg), and producers of phenol through cumene peroxidation (20 kkg). Other sources discharge only to POTW's, have minor aquatic emissions, or there is no information available regarding their discharges. It should be mentioned that discharges to land (comprising 6% of all environmental releases) also have a high likelihood of movement into groundwater or nearby surface waters,

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TABLE 13. GENERAL PHYSICAL PROPERTIES OF PHENOL

Molecular weight	94.11 grams
Melting point	41°C
Boiling point	182°C
Solubility (g/100g H_2O at 25°C)	9.3
Acid dissociation constant (Ka)	1.1×10^{-10}
Vapor pressure at 20°C (torr)	0.5293
Log octanol/water partition coefficient	1.46

Source: Versar 1979a, Morrison and Boyd 1974.

especially at unsupervised landfills or other disposal sites. This particular pathway is described later (see Pathway #5).

Since release to surface water is such a significant source of phenol release to the environment, this pathway is considered here in detail. The section is divided into the following topics:

- Fate processes affecting phenol in surface water (hydrolysis, photolysis and photooxidation, oxidation, volatilization, adsorption, biodegradation, and bioaccumulation);
- Field studies;
- EXAMS model results; and
- Monitoring data.

2. Fate Processes

a. Hydrolysis

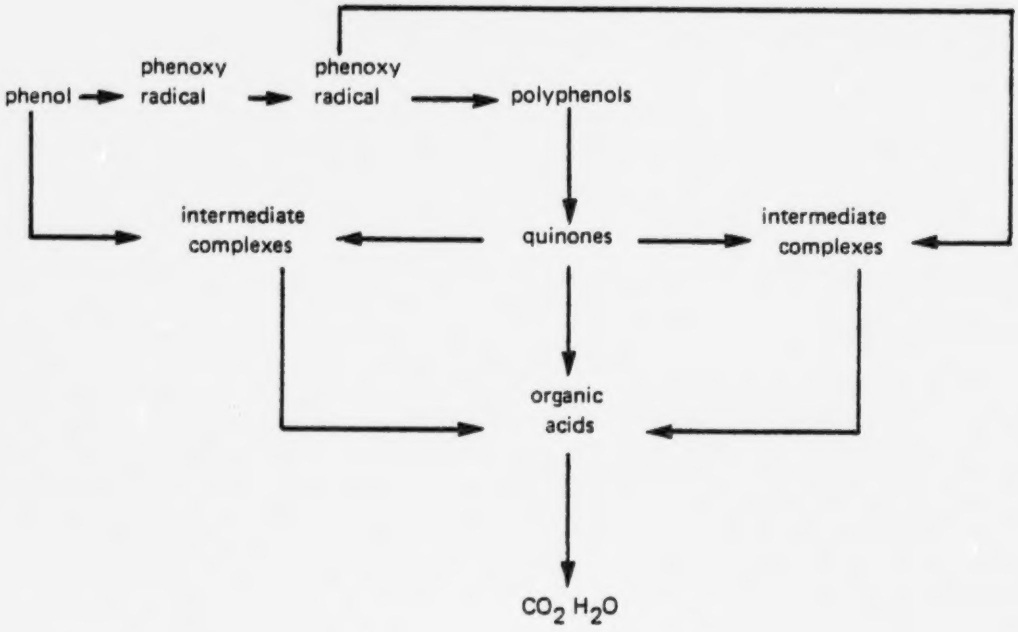
Little information appears to exist concerning the hydrolysis of phenol. By extrapolation of data from similar compounds, however, phenol is assumed to be relatively resistant to hydrolysis. This is because the covalent bond of a substituent attached to an aromatic ring is usually stable due to the high negative-charge density of aromatic nuclei (Versar 1979a). An OH radical rate constant for hydrolysis of phenol in aqueous solution is given as $k = 8.2\text{-}85. \times 10^{-9}/\text{mole}/\text{cm}^3/\text{sec}$ (Shetiya et al. 1976).

b. Photolysis and Photooxidation

Studies of phenol photodecomposition under artificial light indicate a potential for photolysis (Galan and Svith 1976, Joschek and Miller 1966). It is not known how common this fate process is under natural light conditions however the process could be employed in tertiary waste treatment to degrade phenol.

An aqueous solution of phenol with pollutant-level concentrations was passed through a reactor irradiated at 254 nm, not an occurring wavelength in the atmosphere. Phenol was photolyzed and decomposed. Final degradation products included CO_2 and H_2O; intermediates included benzoquinone, predominately as a transient product, as well as some unidentified stable intermediates. The postulated oxidation sequence is shown in Figure 6. The initial activation of phenol to produce the phenoxy radical and the ring opening and conversion of quinones to organic acids are the slowest steps. No rate constants were calculated in this experiment.

In another similar experiment (Joschek and Miller 1966), an aqueous phenol solution was irradiated with a low-pressure mercury lamp at 253.7 nm, a wavelength outside the spectrum of sunlight. The results are


phenol
phenoxy radical
phenoxy radical
polyphenols
intermediate complexes
quinones
intermediate complexes
organic acids
CO2 H2O

Source: Galan and Smith 1976.

FIGURE 6 PHOTOOXIDATION PATHWAYS FOR PHENOL

therefore, not directly applicable to environmental conditions. Certain compounds were identified as degradation products and are listed in Figure 7. The products were compatible with the general categories described in the previous experiment (in Figure 6). No rate constants were calculated from the results of this experiment.

Phenol was also detected as a photodecomposition product in a similar experiment on 4-chlorophenoxyacetic acid in which chlorophenol was an intermediate (Crosby and Wong 1973).

c. Oxidation

Phenol reacts readily with chlorine and bromine in water, forming halogenated compounds (Versar 1979b). Chlorine or ozone can oxidize phenol to hydroquinone and other products (Versar 1979b). These processes are described in greater detail in Pathway #5. No information on the significance of these reactions outside of wastewater treatment was available but it is expected to be low unless the reactants are immediately available (e.g., in the same effluent).

d. Volatilization

No information was found concerning the rate of volatilization of phenol from water except that the rate was expected to be slow based on similar compounds (Versar 1979a). A volatilization half-life for a representative condition can be computed, however, by the following method (Thomas 1980). The Henry's law constant, which relates the concentration in air above a solution to the concentration in the solution, can be found by use of:

$$H' = P_{vp}/S$$

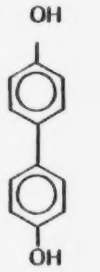
where P_{vp} = vapor pressure of phenol, 7×10^{-4} atm

S = solubility of phenol, 988 moles/m^3

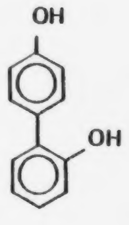
so that H' is $\sim 7 \times 10^{-7}$ atm-m^3/mole. Based on this value, phenol is expected to volatilize very slowly, with the rate controlled by slow diffusion through the air (Mackay 1977, MacKay and Yuen 1979). The half-life for volatilization can be estimated by using the two-film theory of volatilization described by Liss and Slater (Liss and Slater 1974). A phase-exchange coefficient is used to estimate the rate of transfer across the air or water films adjacent to the interface between air and water. Using the liquid-phase exchange coefficient, the half-life is

$$t_{1/2} = 69Z/K_L$$

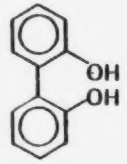
where $K_L = Hk_g \, k_l/(Hk_g + k_l)$, the overall liquid-phase exchange coefficient, cm/hr (Liss and Slater 1974).



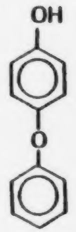
4,4' – Dihydroxybiphenyl



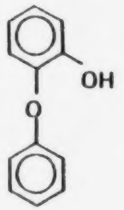
2,4' – Dihydroxybiphenyl



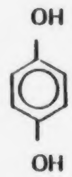
2,2' – Dihydroxybiphenyl



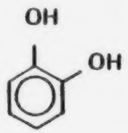
4 – Hydroxdiphenyl ether



2 – Hydroxydiphenyl ether



Hydroquinone



Catechol

Source: Joschek and Miller 1966.

FIGURE 7 PHOTODECOMPOSITION PRODUCTS OF PHENOL

H'/KT = $H'/0.024 = 2.9 \times 10^{-5}$ (R is the gas constant, 8.2×10^{-5} atm-m^3/mole K and T is temperature).

k_g = $1137.5\ (V_{wind} + V_{curr})\ (18/M)^{.5}$, gas-phase exchange coefficient, cm/hr (Southworth 1979)

V_{wind} = wind speed, m/sec

V_{curr} = current speed, m/sec

k_1 = $23.51\ (V_{curr}^{.969} / Z^{.673})\ (32/M)^{.5}\ \exp\ [.526\ (V_{wind}^{-1.9})]$, liquid-phase exchange coefficient, cm/hr (Southworth 1979)

Z = average depth of water body, m

Assuming the following set of conditions; $V_{wind} = 2$ m/sec, $V_{curr} = 1$ m/sec, and $Z = 1$ m, then the computed half-life for volatilization from a water body having these characteristics is estimated to be ~1,550 hours or about two months. An increase in wind speed may reduce the volatilization rate by a factor of three for a shallower water body, but as depth increases the estimated half-life increases to a year or more, even at higher wind speeds.

e. Adsorption

The solid form of phenol is soluble and will sink when spilled and then dissolve. Solutions of phenol (50% benzo-phenol) will sink when spilled and may remain at the bottom of the water body in a concentrated layer in a stratified system. The potential for contact with and sorption on the sediments will therefore be greatly increased in these situations.

Due to phenol's low log octanol/water partition coefficient (1.46), however, adsorption onto sediment is theoretically not expected to be a significant fate process. The relatively low sediment concentrations reported in monitoring data compared to those for other, for example, chlorinated compounds support this hypothesis. (See Monitoring Data section of Pathway #1). The process of adsorption on both organic and inorganic matter is discussed in greater detail in Pathway #5.

The phenol that sinks to the bottom of a water body is subject to the turbulence and mixing of the system it is in as well as to its own rate of diffusion. Therefore, except under conditions of extreme stratification, this phenol would soon be evenly distributed throughout the water body.

f. Biodegradation

Phenol is biodegradable by microorganisms which are prevalent in the natural soil and water environments. Algae, yeasts, bacteria, other microorganisms, broadleaf plants, and higher plants are capable of degrading phenol (Versar 1979a, Versar 1979b, Yang and Humphrey 1975, Vela and Ralston 1978, Throp 1975, Hill and Campbell 1975, Ellis 1977) and will contribute to the removal of moderate amounts of phenolic compounds from the aqueous environment if concentrations of these compounds do not reach toxic levels for these species (see Section V-B).

Microorganisms have developed enzymes capable of breaking down phenol from natural and man-made sources. Nitrogen and phosphate must be available for quick breakdown (Versar 1979b). In stagnant water phenol will support organism growth as a sole carbon source (Versar 1979a). One to two pounds of oxygen per pound of phenol may be utilized by biodegraders in metabolizing dissolved phenol in the first few days of degradation at a rate high enough to deplete local supplies of oxygen (U.S. EPA 1979). In studies using yeasts as the degrading species, large populations of yeast grew on a phenol substrate, oxygen demand was high, and oxygen transfer rather than phenol concentration became the growth-limiting factor (Yang and Humphrey 1975). A mixed culture of algae and microorganisms degrades phenol faster than only heterotrophs since algae supply oxygen to the system (Versar 1979b). Streams may therefore be highly resistant to self-purification below a certain phenol level (Faust et al. 1969).

Phenol concentration affects the growth rate of microorganisms, as well as its own degradation rate. Where phenol is highly diluted, biodegradation may become unimportant since microorganisms apparently will preferentially feed on other material if the phenol concentration is too low (Versar 1979a). At medium concentrations (1-10 mg/l) phenol is more likely to be a metabolic stimulant and be degraded rapidly (Versar 1979b). At higher concentrations, of 100 mg/l or greater (Yang and Humphrey 1975), microorganisms are inhibited or killed so that growth and degradation cease (Versar 1977, Hill and Campbell 1975). Microorganisms have been observed to acclimate to high concentrations of phenol, but fluctuations in phenol concentrations may prevent a constant population level (Versar 1979b). However, phenol can be inhibitory at lower concentrations to these organisms which use it as a growth substrate (Hill and Campbell 1975).

Temperature and pH changes affect the phenol degradation rate (Versar 1979b, Yang and Humphrey 1975). Higher temperatures may increase phenol toxicity to microorganisms (Versar 1979b). In studies of temperature effects on phenol degradation (Vela and Ralston 1978), it was found that the rate of phenol degradation was not significantly influenced by temperatures between 10° and 24°C, but that phenol degradation rapidly fell off at temperatures between 2° and 10°C. This decline in degradation was probably due to a reduction in the numbers and/or activity of the microbial population.

In the biodegradation of phenol, CO_2, H_2O, and biological cells are end products of the aerobic reactions (Throp 1975). Catechol, $C_6H_4(OH)_2$, is a metabolic intermediate in the biooxidation of phenol (Hill and Campbell 1975). A study of biodegradation of phenol by algae (Ellis 1977) reported that algae metabolized catechol more effectively than phenol. This pattern was not unexpected since almost all known enzymatic oxidation reactions capable of aromatic ring fission require at least two hydroxyl groups on the ring and catechol is therefore able to be oxidized immediately.

Some degradation "rates" are known, but these have not been described in sufficient detail to allow them to be applied to environmental situations in general. Table 14 presents a number of these rates measured under different conditions. Phenol (dissolved in water) in a microcosm study degraded at a rate of 2 mg/1/day and in the presence of soil and plants at 3-5 mg/1/day (U.S. EPA 1979 a). It has been degraded at 30 ug/1/hr from an initial concentration of 125 ug/1 in river water (Versar 1979a). At 20°C 1 mg/1 of phenol was assimilated in 1-7 days, and at 4°C the time was 5-19 days (U.S.EPA 1979a). Under anaerobic conditions degradation was even slower (U.S. EPA 1979a).

g. Bioaccumulation

There is a limited amount of published information on the bioaccumulation of phenol by aquatic organisms. In this section, data were examined primarily for fish due to the potential for human exposure through fish consumption. Microcosm, field, or system studies of concentration changes in biota and media over time or of the biomagnification of phenol in the food chain were not available. The lack of field observations to reinforce laboratory findings may be due to problems with field chemical analysis techniques (UNFAO 1973).

i. Variables Affecting Bioaccumulation

The variables that affect bioaccumulation are likely to be the same as those that affect toxicity. The toxicity of phenol to rainbow trout was increased by:

- a decrease in dissolved-oxygen content,
- an increase in water salinity, and
- a decrease in temperature,

but was not affected by changes in pH or water hardness (UNFAO 1973). Another study noted that the environmental factors affecting the toxicity

Table 14. BIODEGRADATION RATES FOR PHENOL

	Rate Constant or Index	Reference
Microcosm study (Water only)	2,000 ug 1^{-1} hr^{-1}	U.S. EPA 1979a
Microcosm study (with soil and plants)	3,000-5,000ug 1^{-1} hr^{-1}	U.S. EPA 1979a
River water population	30 ug 1^{-1} hr^{-1} (initial conc. at 125 ug/1)	U.S. EPA 1979a
CO_2 evolution in river population	80 mg COD g^{-1} hr^{-1}	Pitter 1976
Shake flask with river water population	0.079 day^{-1} ($t_{1/2}$ $\cong$ 9 days)	Lee and Ryan 1979
BOD test with wastewater population	Refractory Index = 0.87* (classified as highly degradable)	Bedard 1976
BOD test with non-specified population	BOD/COD ratio = 0.81	Lyman et al. 1974
Static flask with waste-water population	~100% degradation in 7 days (initial concentration at 5, 10 mg/1)	Tabak et al. 1980

* Based on BOD/COD ratio.

of phenolics include photolytic action, microbial degradation, pH, water hardness, and temperature (Buikema _et al_. 1979). These factors are discussed in greater detail in Section VI-B.

ii. Uptake Rates and Concentrations

Although no information derived from field studies was available, it is worth noting that soluble phenols derived from leaf litter, lignan, and humic acids are present in natural surface waters. Total phenol concentrations ranging from none detected to 0.21 mg/l have been detected in rivers in New Jersey and from 0.006 to 12 mg/l in a reservoir (Buikema _et al_. 1979). These background levels in natural waters suggest that biota are exposed to background levels of phenol as well as to man-made releases.

Table 15 summarizes the accumulated levels of phenol and bioconcentration factors from two studies using goldfish (_Carassius auratus_). The amount of phenol accumulated ("body burden") increased with exposure to higher phenol concentrations. The maximum accumulation after 5 days, 156 ug/g, was measured in fish exposed to 100 mg/l phenol. Kobayashi and Akitake (1975) reported concentrations of phenol in dead goldfish that ranged from 68 to 85 ug/g following exposure to 40 mg/l in water.

Phenol concentrations in fish (33 observations) reported in the STORET data base ranged from non-detectable to as high as 50 mg/kg. The samples consisted of both remarked (at or below the detection limit) and unremarked data. The phenol concentrations in the background water were not available; however no mean major river basin concentration exceeded 1.0 mg/l during the years the tissue concentrations were measured. The reason for such high tissue levels in contrast to the low levels measured under laboratory conditions is not clear. A possible explanation is errors due to the analytical techniques used or due to the way the data were reported in the STORET system. Such high concentrations are expected to be uncommon based on the rapid metabolism and excretion of phenol observed under experimental conditions.

iii. Bioconcentration Factors (BCF)

The summary of bioconcentration factors in Table 15 indicates that phenol is rapidly absorbed by goldfish, showing a concentration factor of 1.64 after one hour of exposure to 15 mg/l phenol, but that the increase in phenol in fish then becomes slower. In general, the BCF's ranged from 1.2 to 2.3. It was assumed that an equilibrium was reached during the exposure period. The BCF appeared to decrease with increasing exposure concentrations (Table 15).

The U.S. EPA estimated a weighted-average BCF for phenol in the edible portions (muscle) of aquatic organisms to be 2.3. This was calculated from an octanol-water partition coefficient of 31 and an adjustment factor of 0.2875 and the equation "$\text{LOG BCF} = 0.76 \text{ Log } P - 0.23$" (U.S. EPA 1979b). This estimation is compatible with the observations described in Table 15.

TABLE 15. AMOUNT OF PHENOL ACCUMULATED IN FISH
(ug/g body weight)

Phenol Concentration (mg/l)	Exposure Time 1 hour	 1 day	 5 days	Unspecified	Reference
unpolluted river				<0.3 (Roach)	UNFAO 1973
2-7 (polluted river)				3.2 (Bream and Barbel)	UNFAO 1973
10		22 (2.2)[1]	14 (1.4)	Goldfish	Kobayashi and Akitake 1975
15	24.7 (1.64)	30.3 (2.02)		"	Kobayashi *et al.* 1976a
20		39 (1.95)	26 (1.3)	"	Kobayashi and Akitake 1975
60		-	92 (1.5)	"	Kobayashi and Akitake 1975
"		96[2] (1.6)	-		
100		-	156 (1.56)	"	Kobayashi and Akitake 1975
"		138[2] (1.38)	138[2] (1.38)		

STORET DATA[3]
1977 ND (mean and max)--2 samples
1978 16.7 (mean); 50 (max)--27 samples
1979 16.0 (mean); 16.0 (max)--4 samples

---

[1] Calculated bioconcentration factor in parenthesis. This is the ratio of tissue level to water level.
[2] Fish found dead in media
[3] EPA (1980d).

iv. Organs Where Phenol is Accumulated

In studies of the distribution of phenol in the organs of five fish species (carp, bream, trout, brown bullhead, and goldfish), it appears that a greater concentration of phenol accumulated in the gall bladder, liver, and other visceral organs than in muscle and gill tissue (see Table 16). Kobayashi (Kobayashi et al. 1976a) found that in goldfish, except for the gall bladder, the phenol concentration in each organ reached an equilibrium after two hours of exposure and rapidly decreased after the fish was transferred to clean water. Only the concentration in the gall bladder increased with exposure time, even after transfer to clean water.

v. Metabolic Effects on Bioaccumulation

Many animal species have the ability to detoxify phenol by the formation of conjugation products with body glucuronides and sulfates (UNFAO 1973). Maickel (Maickel et al. 1958) found that frogs (*Rana pipiens*) excrete 90-95% of absorbed phenols as conjugated compounds 48 hours after parenteral administration. Toads (*Bufo marinus*) and mammals also have the ability to conjugate phenols. Lower vertebrates, such as goldfish, perch, and tadpoles, are considered incapable of forming either glucuronides or sulfates (Maickel et al. 1958).

However, Kobayashi (Kobayashi et al. 1976b) was able to identify two conjugates in goldfish: phenyl-B-glucuronide, which accumulates in the bile, and phenylsulfate, which is excreted directly into surrounding water (see Figure 8). The biliary excretion of the glucuronide is a general detoxication mechanism that fish use for phenolic compounds. The renal excretion of conjugates is considered to be a minor route compared with branchial and biliary excretion (Kobayashi et al. 1976b).

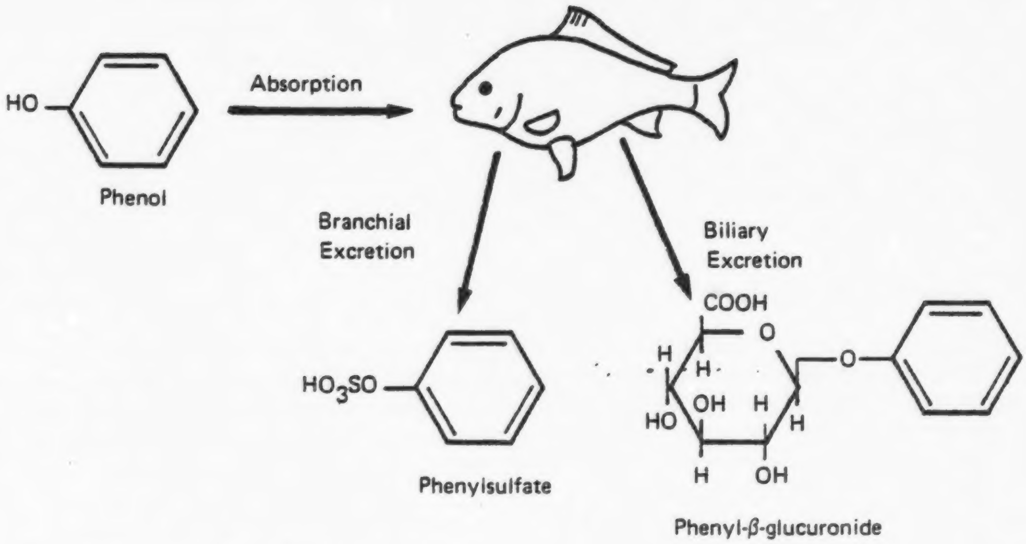
vi. Depuration

Kobayashi and Akitake (1975) found that phenol absorbed by goldfish was so rapidly excreted that the concentration fell to 25% of the initial level just one hour after transfer of the fish to clean water. Subsequent decrease was slower. The authors suggested that the low bioconcentration factors observed reflected a rate of excretion almost equal to the rate of absorption during the beginning of the exposure period.

In carp, phenol elimination was also immediate, with the maximum rate of excretion occurring 10-15 minutes after administration of 200 mg/kg phenol, either orally or by intraperitoneal injection (Buikema et al. 1979). Seventy-five percent of the phenol was eliminated after one hour in clean water, and 85-90% was eliminated after three to four hours. Rainbow trout have a normal urinary excretion rate of 0.7 mg monohydric phenol/kg/day and about 8 mg total phenols/kg/day. Those rates showed a graded increase as the exposure level increased from 1.5 to 6 mg/l (UNFAO 1973). In addition to passive diffusion through the gills and body surface, the biliary excretion of phenol from the liver is an

Table 16. DISTRIBUTION OF PHENOL IN THE ORGANS OF FISH

Species	Exposure to Phenol Time	Concentration	Decreasing Concentration in Organs higher → lower	Reference
Rainbow Trout		10 mg/1	Skin, spleen, liver, kidney, gill (11-25 mg/kg) → muscle (3-2 mg/kg)	UNFAO 1973
Bream	7 days	9 mg/1	Blood & body fluid → cerebral fluid & brain	"
Brown Bullhead	4 days	5 mg/1	Viscera (10 mg/kg) → muscle (6 mg/kg)	"
Carp	3 days	10 mg/1	Liver (19 mg/kg) → gill → kidney → testis → muscle → intestine (7 mg/kg)	"
Goldfish	1 day	15 ppm (~ mg/1)	Gall bladder (114.7 mg/kg) → liver & pancreas (41.1 mg/kg) → spleen (33.9 mg/kg) → testis (26.6 mg/kg) → skin (24 mg/kg) muscle (23.4 mg/kg) → gills (22 mg/kg) scales (18.9 mg/kg)	Kobayashi *et al.* 1976a


HO
Phenol
Absorption
Branchial
Excretion
Biliary
Excretion
HO_3SO
Phenylsulfate
COOH
O
H
H
OH
H
HO
H
H
OH
O
Phenyl-β-glucuronide

Source: Kobayashi *et al.* 1976.

FIGURE 8 A SCHEMATIC VIEW OF THE MAIN EXCRETION ROUTES FOR THE CONJUGATED PHENOLS IN FISH

important mechanism for the depuration of phenol (Kobayashi et al. 1976a).

Although the depuration of phenol from tissue is rapid, a phenolic flavor may persist in fish flesh for several weeks following removal to uncontaminated water (UNFAO 1973).

vii. Biomagnification in Food Chains

The literature did not provide any direct evidence for, or discussion of, differences in bioaccumulation between lower- and higher-trophic level species. However, one study discussed the transfer of phenol to fish, as indicated by the phenolic flavor in the flesh, through ingesting contaminated tubifex worms illustrating movement of phenol up one trophic level in the food chain. In addition, it has been shown that other animals can acquire phenolic flavor by eating "tainted fish (UNFAO 1973). The low bioconcentration factors observed in biota and phenol's relatively short biological half-life indicate that biomagnification in the food chain is probably minimal. No direct evidence on biomagnification was available.

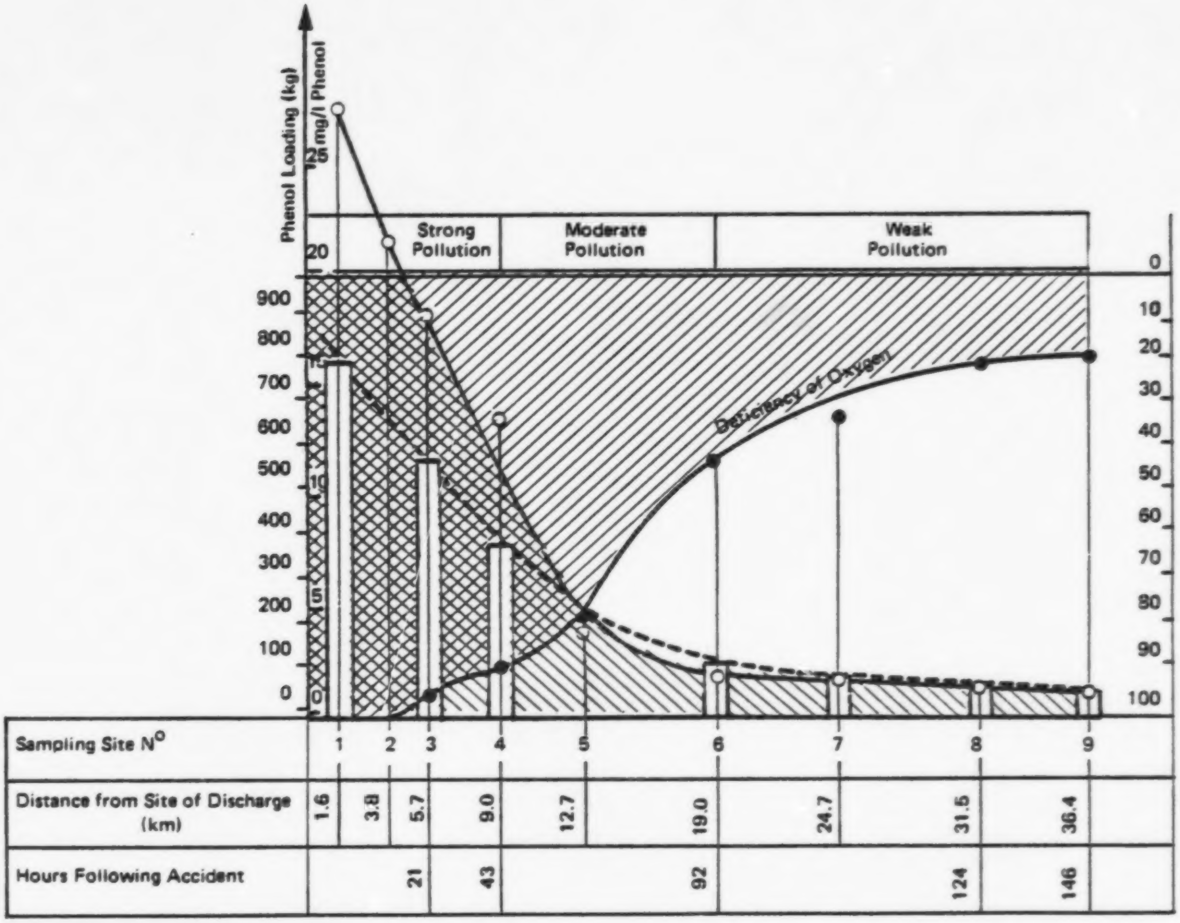
viii. Summary Statement

Studies of the bioaccumulation of phenol were only available for aquatic organisms. Absorption is the primary route of intake. Phenol concentrations of 14 to 136 ug/gram of body weight were reported for goldfish exposed to 10-100 mg/l phenol for 1-5 days. The bioconcentration factors of phenol were low, ranging from 1.2 to 2.3 in goldfish. Phenol tends to accumulate in higher concentrations in the gall bladder, liver, and visceral organs than in the muscle and gills of fish. Higher vertebrates (e.g., mammals) have the ability to detoxify phenol by forming conjugation products with glucuronides and sulfates; fish do not appear to possess this mechanism, excretion of phenol occurs primarily through bronchial diffusion and biliary excretion. No direct evidence was obtained for biomagnification of phenol; however, it does not appear to be significant.

3. Field Studies

The following section describes in detail several situations involving phenol discharges to aquatic systems. All of the studies are concerned with generic phenol wastes and not phenol specifically; however, since phenol is present in these wastes, their study gives some insight into the environmental problems related to phenol. The studies characterized the sources and concentrations of typical discharge practices and, in one case, of a spill. Table 17 briefly presents the results of each study. The following text discusses the conclusions more thoroughly.

In one discharge situation, a very large amount of phenol (11,000 kg of primarily monohydric phenols) from a phenol-producing plant was spilled from a holding tank at high concentrations (800-1,000 mg/l) (see Table 17). Approximately 10% (1,100 kg) entered a nearby river within a half-hour period. Figure 9 depicts the relationship between maximum phenol levels measured and oxygen



Sources: Krombach and Barthel, 1964.

FIGURE 9 MAXIMUM CONCENTRATIONS AND LOADS OF PHENOL FOLLOWING RELEASE INTO RIVER

Table 17. FIELD STUDIES OF PHENOL DISCHARGES TO AQUATIC SYSTEMS

<table>
<tr><th>Phenol Source</th><th>Aquatic System</th><th>Observations</th><th>Reference</th></tr>
<tr><td>Phenol-producing factory (benzene sulfonation)</td><td>River in Luxembourg</td><td>A holding dike broke releasing 11,000 kg of phenols (80-1,000 mg/l in waste). Of this, 90% (9,900 kg) was retained in flooded soil and 1,100 kg entered the river within 1/2 hour. Samples were taken in a 36-km stretch. Results were as follows:
<table>
<tr><th>Distance Downstream (km)</th><th>Max. Conc. of Phenols (mg/l)</th><th>Load of Phenols (kg)[1]</th><th>Max. Oxygen Deficit (%)</th></tr>
<tr><td>1.6</td><td>28</td><td>810</td><td>100</td></tr>
<tr><td>3.8</td><td>22</td><td></td><td>100</td></tr>
<tr><td>5.7</td><td>18.3</td><td>532</td><td>94</td></tr>
<tr><td>9.0</td><td>14.0</td><td>396</td><td>88</td></tr>
<tr><td>12.7</td><td>3.8</td><td></td><td>76</td></tr>
<tr><td>19.0</td><td>2.05</td><td>126</td><td>41</td></tr>
<tr><td>24.7</td><td>2.0</td><td></td><td>32</td></tr>
<tr><td>31.5</td><td>1.4</td><td>80</td><td>19</td></tr>
<tr><td>36.4</td><td>1.0</td><td>60</td><td>20</td></tr>
</table>
[1] Based on flow rate and phenols concentration in each river segment</td><td>Krombach and Barthel 1964</td></tr>
<tr><td>Petroleum refining complexes and petrochemical plant (cumene peroxidation)</td><td>St. Lawrence River</td><td>Background upstream phenols level = 2-10 ug/l mean (summer and winter). Downstream phenols level ranged from equivalent to background up to 100 ug/l. Means were as follows:
<table>
<tr><th>Season</th><th>Distance from Last Source</th><th>Conc. (ug/l)</th><th>Mass Balance (kg/day)</th></tr>
<tr><td>Winter (0.1° C water)</td><td>3,000 m downstream</td><td>21.2</td><td>2,330</td></tr>
<tr><td></td><td>5,200 m downstream</td><td>24.2</td><td>2,650</td></tr>
<tr><td></td><td>7,300 m downstream</td><td>17.2</td><td>1,940</td></tr>
<tr><td>Summer (17-21°C water)</td><td>3,000 m downstream</td><td>4.0</td><td>560</td></tr>
<tr><td></td><td>5,800 m downstream</td><td>2.0</td><td>275</td></tr>
</table></td><td>Polisios et al. 1975</td></tr>
<tr><td>Rural and urban areas</td><td>Six river systems in New Jersey</td><td>Phenols samples ranged from not detectable to 210 ug/l</td><td>Faust et al. 1969</td></tr>
</table>

deficiency, distance from plant, and time since event. At 12.7 km downstream and approximately 63 hours (2 1/2 days) following the event, the phenol concentration began to level off (at approximately 5 mg/l). The background phenol concentration resulting from normal discharge practices (preceeding the incident) was 1 mg/l. The maximum phenol level measured following the incident was 28 mg/l at 1.6 km downstream and at approximately 6 hours after the event. The phenols load fell from 810 to 60 kg in 6 days, a 93% decrease. The accompanying deficit in oxygen levels was attributed to oxidation of phenols and, initially, other waste products present (sulfites).

The authors also calculated a decay rate constant for each sampling point along the river as a function of flow rate. The decay rate (k) at 15°-16°C varied from 0.13 day^{-1} (at >30 km) to 0.23 day^{-1} (within 6 km). The average k was 0.20 day^{-1} which the authors claimed was comparable to a wastewater treatment plant decay rate for organic constituents. The rate constant of 0.20 day^{-1} results in a half-life (based on first-order kinetics) of 3.5 days.

In a Canadian study of the fate of phenols discharged to river water (Polosios _et al_. 1975) Polosios found a significant difference (up to one order of magnitude) between winter and summer mean concentrations of phenols downstream (see Table 17). Possible explanations offered included seasonal changes in waste products from the refinery (oil used in winter, gas in summer), crude oil feedstock differences, and a lower rate of microbial degradation during the winter. Considering the mass balance of phenols in the river (mean concentration x stream flow in several segments), again in the summer a decrease in load was evident further from the plants while no decrease was present in winter.

The rate of phenol disappearance was estimated from the summer observations and compared to both laboratory-derived rates and another field study (see Table 18).

TABLE 18.

RATE OF PHENOL DISAPPEARANCE FROM RIVER WATER[1]

	Reaction Rate (ug l^{-1} hr^{-1})	Reaction Rate (umole l^{-1} hr^{-1})	Temperature (°C)	Mean Concentration (ug/l)
Field[2]	1.3	0.014	17.7-20.7	3.0
Lab	2	0.021	23	15
Field[3]	0.09-0.17	0.0010-0.0018	15-16	15

---

[1] Assuming first-order kinetics apply to ug/l range.

[2] Polisios et al. 1975; [3] Kromach and Barthel 1964.
Source: Arthur D. Little, Inc. estimates
recalculated from Krombach and Barthel 1964

In a monitoring survey of phenols in six streams in New Jersey (Faust et al. 1969), concentrations in industrial, agricultural, urban, and rural areas were measured. A 24-hour sampling survey was conducted at one station which receives industrial and municipal wastewaters. The maximum levels of phenols (66 ug/l) occurred between 8:45 AM and 4:45 PM, dropping to 12 mg/l (see Figure 10). In addition, as the stream flow increased so did the phenols concentration until approximately 2:00 PM; then it dropped significantly. Phenols concentrations in a relatively unpolluted river ranged from ∿6 ug/l to 13 ug/l. In an agricultural area they ranged from 4-10 ug/l. Industrial areas had considerably higher levels, up to 210 ug/l. The study did not characterize the types of industries contributing to high phenols concentrations nor did it distinguish between industrial and domestic contributions.

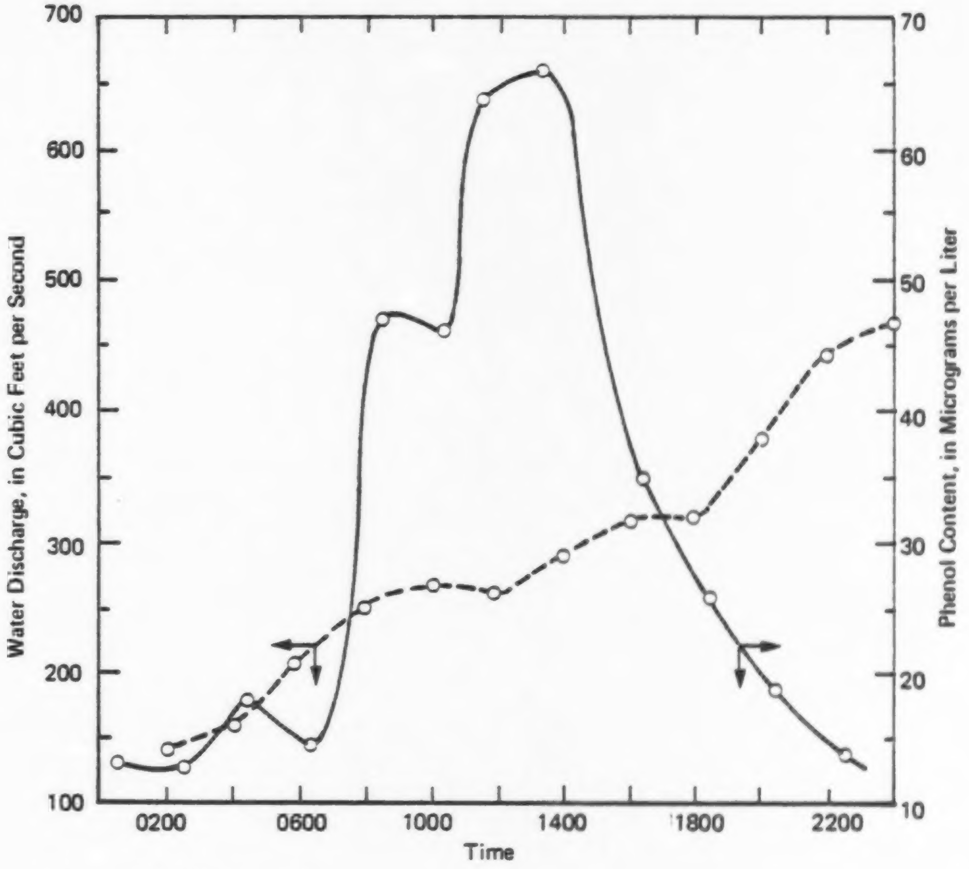
4. EXAMS Model Results

The EXAMS model (U.S. EPA 1980a) was implemented for purposes of estimating phenol concentrations in water and other aquatic media and to better understand the significance of fate processes in determining phenol concentrations. Simulations were conducted using the data base for phenol estimated by Stanford Research Institute (SRI 1980). The primary processes determining phenol's persistence were biodegradation and chemical oxidation. The results of the simulation including phenol concentrations, total accumulation, and percentage loss by selected means are presented in Table 19.

According to the data entered, at an equilibrium loading rate of 3 kg/hr a half-life of 10 minutes was predicted in both a eutrophic lake and a 1-km turbid river segment with resulting maximum water-column concentrations of 3 ug/l and 1.3 ug/l, respectively. These results are consistent with the surface water concentrations (means) reported in the STORET monitoring data base (see Section IV-C-4) since 1972; these ranged from 1.0 to 52 ug/l; the variance between the two sets of numbers was greater than a factor of 40.

To determine how meaningful these comparisons are, the loading rates of phenol to water in various industrial effluents can be examined. Presentation of these effluent data must be accompanied by the warning that the effluent concentrations and flow rates are derived from a limited number of samples. It is not possible, therefore, to know how representative the estimated discharge rates are of each industry as a whole. They serve only as examples of industrial loading rates to water. Table 20 presents the estimations based on Effluent Guidelines sampling data presented in Appendix A.

As can be seen in Table 20, most of the estimated loading rates are less than the 3-kg/hr rate used in the EXAMS analysis. Only the iron and steel byproduct coking category was higher. None of these loadings would be significant enough to result in EXAMS-estimated water-column concentrations greater then 5 ug/l in the stream and lake environment.



Source: Faust *et al.* 1969.

FIGURE 10 VARIATIONS IN PHENOL CONTENT AND STREAM FLOW DURING 24-HOUR SURVEILLANCE

TABLE 19. RESULTS OF EXAMS SIMULATION

Maximum Phenol Concentration (mg/l)

	Water Column (diss.)	(Total)	Bottom Sediment	Plankton ug/g	Benthos ug/g	Total in Sediment (dry) (mg/kg)	Total Steady State Accum. (kg)
Turbid River $3.0\ \mathrm{kg\ hr^{-1}}$ Loading	$2.97\mathrm{x}10^{-3}$	$2.97\mathrm{x}10^{-3}$	$1.99\mathrm{x}10^{-4}$	$1.45\mathrm{x}10^{-2}$	$9.68\mathrm{x}10^{-4}$	$1.34\mathrm{x}10^{-4}$	2.7
Eutrophic Lake $3.0\ \mathrm{kg\ hr^{-1}}$ Loading	$1.33\mathrm{x}10^{-3}$	$1.33\mathrm{x}10^{-3}$	$5.63\mathrm{x}10^{-7}$	$6.39\mathrm{x}10^{-3}$	$2.74\mathrm{x}10^{-6}$	$5.30\mathrm{x}10^{-7}$	1.0

	% in Water Column	% in Bottom Sediment	% Lost by Processes				Self-Purification Time (hrs)
			Chemical Transformation	Biological Transformation	Volatilization	Other	
Turbid River	99.95	0.05	0.88	1.33	0.01	97.79	3.34
Eutrophic Lake	100	0	0.33	99.63	0.00	0.04	4.61

In 12 hrs lost 100% of initial concentration in water; 28.14% of sediment.

In 12 hrs lost 99.99% of initial concentration in water; 99.99% of sediment.

Source: U.S. EPA 1980a.

TABLE 20. ESTIMATED LOADING RATES OF PHENOL TO SURFACE WATER FOR SELECTED INDUSTRIAL PLANTS

Direct Dischargers	Discharge Rate (kg/hr)[1]
Petroleum Refiners	1.1
Hardboard[2] S2S	0.2
Insulation - Thermochemical Pulp & Refining[2]	0.1
Hair Pulp, Chrome Tan Retan - Wet Finish[3]	0.4
Hair Save, Chrome Tan Retan[3]	0.1
No Beamhouse[3]	0.4
Iron and Steel: Byproduct Coking	3.9
Iron and Steel: Cold Rolling	0.9
Steam Electric - Ash Handling	1.6[4]

[1] Assuming discharge on 250 days/year for 8 hours/day except where noted.

[2] Subcategory of timber products processing industry.

[3] Subcategory of leather tanning industry.

[4] Assuming discharge on 365 days/year.

Source: Based on Effluents Guidelines data presented throughout Appendix A.

In Table 21, loading rates were estimated from materials balance data (Section III) for two significant aquatic dischargers: phenolic resin producers and phenol producers by cumene peroxidation. Both of the rates were higher than the EXAMS-run loading rate of 3 kg hr^{-1}, especially the rate for phenolic resin producers. Assuming that most of the phenolics discharged is phenol, and that the EXAMS model still exhibits a linear relationship between loading rate and resulting water-column concentration, a phenolic resin producer load would produce concentrations of 43 ug/l and 19 ug/l in the turbid river and eutrophic lake systems, respectively. These levels are not significantly higher than those at the lower loads. Some of the constituents of the phenols waste, however are probably much more persistent than phenol itself, so these results cannot be taken as representative of the fate of phenolic resin waste.

5. Monitoring Data

A limited data base was available describing phenol concentrations in surface water and industrial effluents. Records of phenol concentrations in surface waters in the STORET water quality system (U.S.EPA 1980d) entered since 1978 contain less than 600 remarked and unremarked observations. Records for phenol in sediment and tissue are much lower, roughly 30 observations each. Of the 18 major river basins in the continental-United States, phenol concentrations have been reported in thirteen of them. These basins are located throughout the United States--Northeast, North Atlantic, Southeast, Tennessee River, Ohio River, Lake Erie, Lower Mississippi, Colorado River, California, Pacific Northwest, Great Basin, Lake Huron and Hawaii.

STORET data were far from complete; out of the 555 total observations reported, 514 were remarked data. This means that, in most cases, the value reported is a detection limit and the actual concentration may be below this level. Therefore, remarked and unremarked data were considered separately. Table 22 presents data for U.S. ambient surface water for 1978 through 1980.

Given the limited nature of the data, conclusions concerning phenol levels in the environment should be considered only approximate. The data indicate that the criterion of 300 ug/l recommended by the Environmental Protection Agency (U.S. EPA 1980b) to prevent taste and odor effects has not been violated by major river basin levels since 1978, with the exception of the Tennessee and Ohio River Basins (unremarked data). The major values for these two river basins are based on very limited data, one and nine observations, respectively. Each basin had only one observation which exceeded the criterion level in the last 3 years.

Although a few studies report monitoring levels for waterways, data on phenol levels in various industrial effluents are available. Jungclaus examined both the wastewater and receiving water in the vicinity of a specialty chemicals manufacturing plant and reported finding levels of 0.01 to 0.30 mg/l of phenol in the wastewater, 0.01 to 0.10 mg/l in the river water, and no detectable levels in the sediment. These results have an

TABLE 21. ESTIMATED INDUSTRIAL LOADING RATES OF PHENOL TO SURFACE WATER

Discharger	Loading Rate	Assumptions
Phenolic resin producers	43 kg hr^{-1} (of phenols)	1. 288,020 kkg produced annually 2. Approximately 100 plants 3. 2,880 kkg produced per plant annually 4. 36,400 kg phenol discharged per plant annually (based on a release rate of 30 kg phenol/kkg phenolic resins) 5. 350 kg discharged per day (250 days/year) 6. 43 kg discharged per hour (8 hrs/day)
Cumene producer	7 kg hr^{-1}	1. 1,108,850 kkg produced annually 2. 6 plants 3. 184,800 kkg produced per plant annually 4. 14,000 kg phenol discharged per plant annually (based on a release rate of 0.0755 kg phenol lost/ kkg phenol produced) 5. 56 kg discharged per day (250 days/year) 6. 7 kg discharged per hour (8 hrs/day)

Source: Section III and Appendix A.

TABLE 22. PHENOL CONCENTRATIONS (TOTAL; REMARKED AND UNREMARKED) IN U.S. AMBIENT SURFACE WATER FROM 1978 THROUGH 1980 (µg/l)

	Remarked Data						Unremarked Data					
Major River Basin	No.[1]	Mean	Median	85%[2]	Max	S.D.[3]	No.	Mean	Median	85%	Max	S.D.
Northeast	27	0.07	-[4]	-	1.0	0.3	9	10.6	1.4	14	65	21.1
North Atlantic	36	3.7	2.0	5.0	10.0	2.2	3	4.1	0.3	7.0	7.0	3.5
Southeast	111	194.8	10.0	20.0	10000.0	1334.3	4	54.0	22.0	140.0	140.0	58.7
Tennessee River	36	20.8	25.0	25.0	25.0	6.8	1	6794.0	6794.0	-	6794.0	-
Ohio River	77	6.1	5.0	10.0	10.0	3.6	9	659.5	1.5	26.0	5900.0	1965.2
Lake Erie	1	50.0	50.0	-	50.0	-	-	-	-	-	-	-
Lower Mississippi	51	18.4	10.0	10.0	400.0	54.8	-	-	-	-	-	-
Colorado River	19	33.7	50.0	50.0	50.0	19.8	-	-	-	-	-	-
Pacific Northwest	117	11.8	10.0	25.0	25.0	8.2	12	21.4	0.07	1.7	252.0	72.6
California	9	22.8	15.0	50.0	50.0	16.0	-	-	-	-	-	-
Great Basin	11	35.0	50.0	50.0	50.0	17.9	-	-	-	-	-	-
Hawaii	5	42.0	50.0	50.0	50.0	17.9	-	-	-	-	-	-
Lake Huron	-	-	-	-	-	-	3	0.004	0.002	0.006	0.006	0.002
Gross Analysis	514	52.2	10.0	25.0	10000.0	622.7	41	324.6	1.3	26.0	6794.0	1385.1

[1] Number of observations. [2] 85th percentile. [3] Standard deviation. [4] No observations.

Source: U.S. EPA (1980d)

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estimated error of 20%. Faust (Faust et al. 1969) surveyed total phenols in six New Jersey river basins and found levels ranging from non-detectable to 0.21 mg/l.

Various other miscellaneous reported concentrations in effluents include measurements from three monitoring studies of oil refinery wastewaters with levels ranging from 10 to 100 mg/l. Pitt (Pitt et al. 1975) reported concentrations between 0.006 to 0.012 mg/l in primary sewage plant effluents. Table 23 lists phenol concentrations in the effluents of selected industries.

Concentrations of phenol in sediment have been recorded in major river basins from 1978 to 1980. With 38 observations, it is impossible for the data to be aggregated in a meaningful way for national representation. Average concentrations for the three regions over the three-year period ranged from none-detected to 454 mg/kg for phenol in sediment. These figures are presented in Table 24 along with maximum and minimum values.

D. PATHWAY #2. EMISSIONS TO AIR

1. Introduction

An estimated 64% (12,121 kkg) of all environmental releases of phenol are to air (see Table 2). Emissions during residential and automobile fuel combustion contribute approximately 81% (9,500 kkg) of total air releases; the other significant air source is the phenol producers (by cumene process) which contribute approximately 14% of air releases. The remaining releases to air (totalling ~1,000 kkg) can be attributed to numerous consumptive processes such as production of phenolic resins, bisphenol, and nonylphenol, and losses during transport and storage. Another source which could not be quantified for this study is the combustion of fossil fuel by power plants and other fuel consumers.

Losses to the air are associated with two categories: 1) loss of phenol in steam or vapor from evaporators, coolers, and combustion of fuel (all usually involving high temperatures); and 2) loss of phenol during normal handling and transport. No information was available on handling and transport to determine the amount lost in particulate form from solid phenol cakes and the amount lost in vapor form from liquid.

2. Fate Processes and Field Studies

Given the major sources of phenol releases to air (see Table 2) it can be assumed that most of the chemical releases are in vapor form. In the atmosphere phenol may be adsorbed by airborne particulates. This was indicated by its detection in particulate form as a secondary organic pollutant in an urban atmosphere (Cronin et al. 1977). It is possible, however, that its presence in this situation was due to a phenol-generating reaction involving a parent pollutant (e.g., alkene, alkane) already sorbed on the particulate matter.

TABLE 23. EFFLUENT LEVELS OF PHENOL IN INDUSTRIAL WASTEWATER

Industry	Type	Concentration (mg/l)	Reference
Kraft paper mill (A)[1]	final effluent	ND[2]	Keith 1976
Kraft paper mill (B)[1]	final effluent	ND	Keith 1976
Paper mill	raw waste	10-2,000	Nebel et al. 1976
Petroleum refinery (A)[1]	final effluent	0.88	Baird et al. 1976
Petroleum refinery (B)[1]	final effluent	3016	Baird et al. 1976
Petroleum refinery	8-hour lagoon effluent	0.2	Webb et al. 1973
Integrated oil refinery	raw effluent	120	Volesky et al. 1974
Petrochemicals	fire-dry lagoon effluent	0.06	Webb et al. 1973
Coal gasification	processed effluents	<0.2	Klemetson 1976
Specialty chemicals manufacture	wastewater	0.1-0.3	Jungclaus et al. 1978
Specialty chemicals manufacture	river water	0.01-0.1	Jungclaus et al. 1978
Kraft paper mill	final effluent	0.14-2.4	Fox 1976
Dye manufacturing plant	raw effluent	110-190	Buikema et al. 1979

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[1] Separate plants

[2] Non-detectable

Source: Buikema et al. 1979

TABLE 24. CONCENTRATION OF PHENOL IN SEDIMENT (in mg/kg; 1978-1980)

	Number of Observations	Mean	Median	85%	Max.	S.D.
Remarked Data	24	14.2	0.05	1.0	143	39.1
Unremarked Data	14	102	0.5	208	454	155

Source: U.S. EPA 1980c.

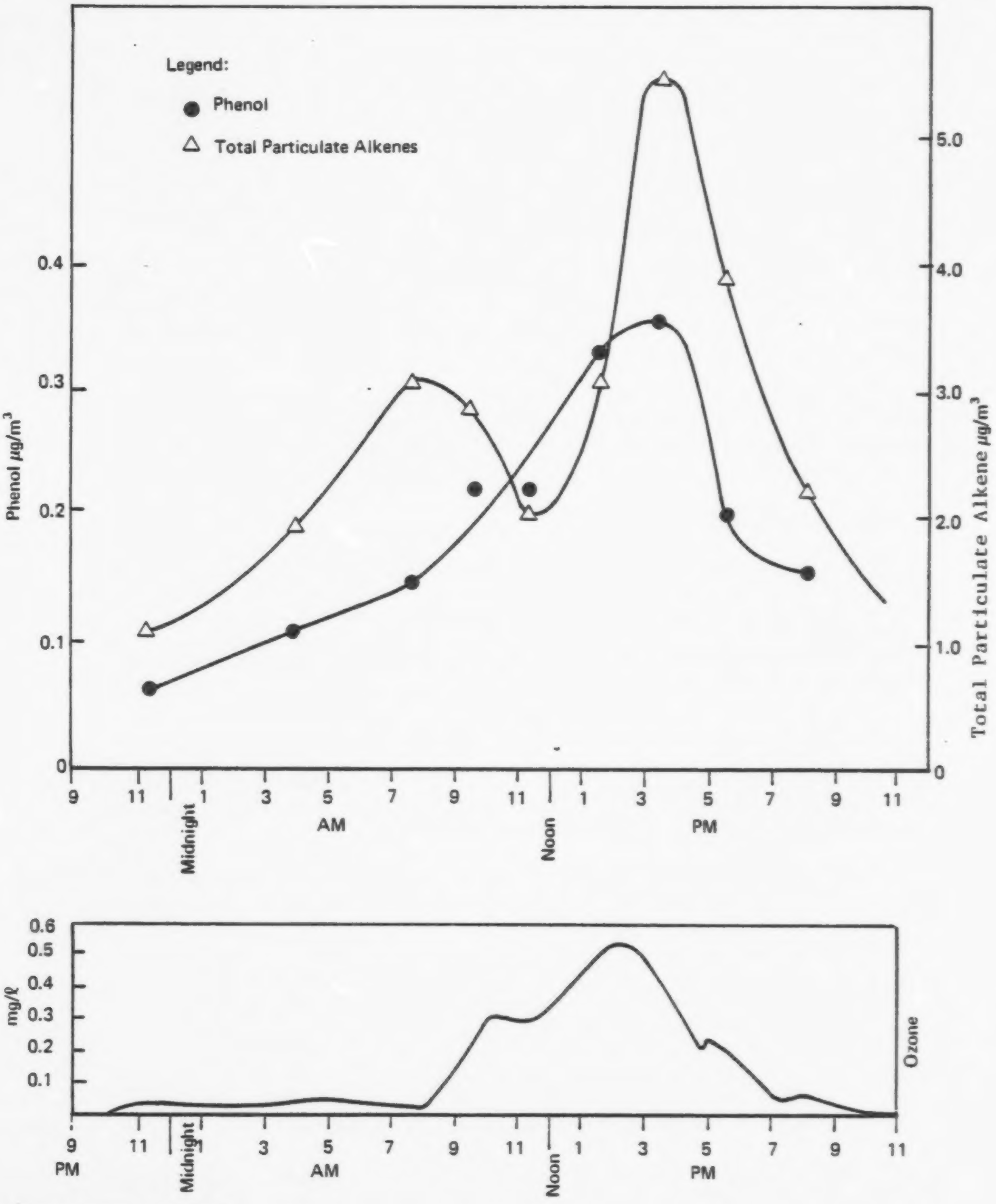
Phenol emissions to the atmosphere are related to combustion of fossil fuels and are therefore higher in urban areas. During periods of smog, phenol is detected as a secondary pollutant or originating from reactions involving primary, directly emitted substances such as total particulate alkanes and alkenes, alkyl benzenes, naphthalene, alky piperidenes, and alkyl nitrites, among others (Cronin et al. 1977). Phenol was one of a group of secondary pollutants characterized by a low vapor pressure and undergoing condensation to form particulates.

The results of monitoring phenol concentrations over a day are presented in Figure 11. Phenol levels were plotted against ozone and total particulate alkene concentrations. Phenol peaks occurred during the same time periods in which ozone peaks were found as well as during periods of maximum sunlight intensity. The phenol peak lagged by 7 hours behind the first alkene peak, suggesting an alkene reaction rate (leading to phenol) on the order of 7 hours. The phenol level reached a peak of approximately 0.35 ug/m^3 at 4:00 PM and rapidly dropped to its daily low of about 0.05 ug/m^3. Destruction of phenol was not attributed to any specific process but may have been due to photooxidation or indirect photolysis (as described later).

In another study of an urban atmosphere conducted in Frankfurt, Germany, phenol concentrations (in addition to CO and lead concentrations) were reported to be related to traffic flow and volume and attributed to the combustion of gasoline (presumably due to breakdown of benzene) by motor vehicles (Deimal and Gableske 1973). Measurements were made every half hour between 6:00 AM and 8:00 PM. Concentrations ranged from <0.020 to 0.289 mg/m^3. Approximately 50% of all observations (total = 402) were ≤ 0.029 mg/m^3 and 75% were ≤ 0.049 mg/m^3. The higher concentrations were associated with periods of inclement weather (presumably inversions). No supporting data were given to substantiate the authors' statement of a relationship between phenol levels and traffic volume. Additionally, no information was given on the possible chemical reactions responsible for reduction of phenol levels following traffic slowdown or on the products of these reactions.

The reason that the German atmospheric levels (Deimal and Gableske 1973) were two to three orders of magnitude higher than the U.S. levels (Cronin et al. 1977) was not obvious but may have been due to differences in the total fuel consumption/combustion between the two cities studied, the distance from sources, or differences in the placement of monitoring equipment.

The two fate processes influencing phenol concentrations in the atmosphere are indirect photolysis and photooxidation (Versar 1979a). Versar estimated, based on interpolations from photodegradation data for m-xylene and toluene, an atmospheric lifetime of a few days for phenol due to photolysis and/or photooxidation of the metastable oxygen-phenol charge transfer complexes.



Source: Cronin *et al.* 1977.

FIGURE 11 PHENOL, ALKENES, AND OZONE VARIATION DURING A HEAVY SMOG EPISODE

Phenol most commonly occurs in the atmosphere in the non-ionic, protonated form, as indicated by its pKa of 10.02 (Versar 1979a). The spectral absorption curve for non-ionic phenol has had peaks measured between 210 and 260 nm (Galan and Smith 1976) and a λ_{max} between 270 and 290 nm (Herrington and Kynaston 1957). Natural sunlight with an ultraviolet sorption spectrum at $\lambda > 290$ nm in the atmosphere is thus not likely to directly photolyze phenol. The anion of phenol has a higher absorption maximum between 287 and 310 nm which does fall within the spectral range of natural sunlight; however, this form is expected to be quite uncommon. Coordination of the phenol oxygen atom with low valence metal cations (e.g., in soil) may increase the ionization of the phenolic proton (Versar 1979b).

Indirect photolysis depends on the presence of nitrosyl, hydroxyl, and alkyl peroxyl radicals, which are generated through the photochemical reactions of nitrogen oxide, water, and organics in the environment (Versar 1979b).

3. Monitoring Data

The only monitoring data available on phenol concentrations in air were those observations discussed in the previous section. To summarize, in two urban areas phenol levels ranged from a low of 0.05 ug/m^3 to 0.35 ug/m^3, fluctuating on a daily basis (Cronin et al. 1977) and from <20 ug/m^3 to 289 ug/m^3 with 50% of all observations less than 30 ug/m^3 (Deimal and Gableske 1973).

E. PATHWAY #3. AIR TO WATER/SOIL: RAINOUT

Following the release of phenol to air, the amount not subject to photodecomposition will be available for transport from the atmosphere to other media through the process of rainout.

If the concentration of phenol in air is known, the nondimensional Henry's law constant can be used to provide an estimate of the concentration in rain. The equation

$$c_{rain} = c_{air}/H^*$$

where: c_{rain} = concentration in rain

c_{air} = concentration in air

H^* = nondimensional Henry's law constant

shows this relationship. Using the nondimensional Henry's law constant of 3×10^{-5} and assuming phenol concentrations of between 50 and 350 ng/m^3 in air (Cronin et al. 1977), the initial concentration in rain will be between 0.4 and 3 mg/l.

These levels will be the initial amount. As rain keeps falling it will cleanse the atmosphere in the immediate vicinity and the concentration will decline. Phenol has been detected in rainfall, but no levels have been quantified (Versar 1979a). Detectable phenol levels in rain can be expected in areas of heavy atmospheric emissions from autos or industries (Versar 1979a).

Only non-quantified observations reported elevated phenol concentrations in industrial-area rain as compared to rural-area rain in Bulgaria (Kurchatova and Mladenova 1975) and detectable levels in snow in an industrial region of Russia (Bobkov 1974).

F. PATHWAY #4. FATE IN POTW'S AND WASTEWATER TREATMENT

1. Introduction

Phenol is frequently detected in the influent of Publicly Owned Treatment Works (POTW's) at concentrations of 1-200 ug/l (Burns and Roe 1980). According to the materials balance on phenol (Section III), the only discharges to POTW's that have been quantified (1,010 kkg annually) originates from phenol resin producers, bisphenol A producers, cumene production facilities, petroleum refineries, timber products, leather tanning, textile, and iron and steel industries. For most of the significant sources of phenol releases to water, no information was available to discriminate between direct and indirect dischargers. Some of the influent load may be attributed to leaching or runoff of phenol into storm sewers, due to the chemical's mobility; however, it is likely that other industries discharge phenol in their effluents to POTW's. Quantification of this contribution will require further investigation.

Phenol can be degraded in wastewater treatment plants by numerous and diverse processes: through biodegradation in activated sludge and trickling filters (Versar 1979a) and removal by chemical treatment (Versar 1979a). Observations at POTW's demonstrate the success of phenol removal by conventional wastewater treatment.

2. Biological Degradation

The biodegradation process for phenol is fast, with a rate on the order of 0.013-7.6 ug/l/hr (Burns and Roe 1980) measured in several studies (Versar 1977). Not all organisms existing in biological process tanks can degrade phenol at the same rate, however. Acclimation is an important factor in achieving consistently good phenol removal in biological treatment systems (SCS 1979, Haller 1978). Removal efficiency is a function of system design. Contact time, flow rate, aeration rate, etc. are key variables in ultimate removal efficiencies. Also the importance of the chemical makeup and variability of the wastewater cannot be overlooked.

At phenol concentrations of between 1 and 10 mg/l most of the phenol will be degraded (Versar 1979a). Oxygen uptake is inhibited by phenol concentrations of between 10 and 100 mg/l. Microorganisms can continue to degrade phenol at concentrations of up to 500 mg/l if the population is allowed time for acclimation. In completely mixed suspended-growth systems using activated sludge or aerated lagoons with long hydraulic retention times, inlet concentrations of 200-300 mg/l were degraded to 0.2-0.5 mg/l (>99%) in about 24 hours (Throp 1975). Oxygen was supplied by mechanical agitation and about 2.5 kg O_2 were consumed per kg of phenol at 30°C. In wastewater treatment of phenol by biological methods, pH must be kept between 6 and 9.5; nitrogen and phosphorous nutrient loadings in the range of BOD_5:N:P = 100:5:1 are optimum for biodegradation. Phosphorous can be added at a concentration ratio of P:phenol = 1:30 to increase the process efficiency. A temperature of 18-35°C is desirable. Under these conditions and in the presence of 0.16 kg phenol per kg of biological solids, biological oxidation was observed to reduce the phenol level of 100-800 mg/l to 1 mg/l. Shock loads of phenol must be monitored and kept below 500 mg/l to prevent a decrease in the efficiency of the system (Throp 1975). Following these shock loads, different steady states may be achieved in the activated sludge reactor due to the lag times for recovery from the shock loads (Pawlowsky *et al*. 1973). Even when acclimated to high concentrations of phenol, activated sludge may have lag times of from about 10 hours at an initial concentration of 200 mg/l, to as high as 7 days at an initial concentration of 700 mg/l before the biodegradation rate becomes rapid.

A trickling-filter biotreatment process can reduce phenol up to 280 mg/l with 99.9% efficiency (Throp 1975). To demonstrate the efficacy of acclimated bacteria on phenol removal, several industrial wastes containing phenol were treated by adding a freeze-dried PHENOBAC® culture (acclimated to phenol) to activated sludge/biological filter processes. Removals were 95%, 99+%, and 99+% for influent phenol concentrations of 86.4, 880, and 332 mg/l, respectively (SCS 1979).

3. Chemical Treatment

Phenol is degraded by a number of chemical treatment methods available for wastewater treatment. Although many of these methods have only a limited distribution, some of them are discussed in the following section. These are chlorination and treatment with hydrogen peroxide, potassium permanganate, ozone, and iron ferrate.

During normal wastewater treatment and not under the special conditions necessary for chlorine-mediated oxidation, it is very common for phenols to become chlorinated to form lower chlorinated phenols. This is discussed separately under Pathway #6.

Chlorine can be used to degrade phenol. At a ratio of 100:1 Cl:phenol, phenol at 123 ug/l is degraded by 12 mg/l of chlorine. Phenol at 13.5 mg/l completely consumes all applied chlorine up to 16.2 mg/l within a contact time of one-half hour (MCA 1972).

The reaction progresses rapidly in the first 15 minutes, followed by a decreasing rate up to 2 hours. With treatment by chlorine at 36 mg/l, the final pH is above 8.4, outside the pH limits for formation of chlorophenol (Throp 1975).

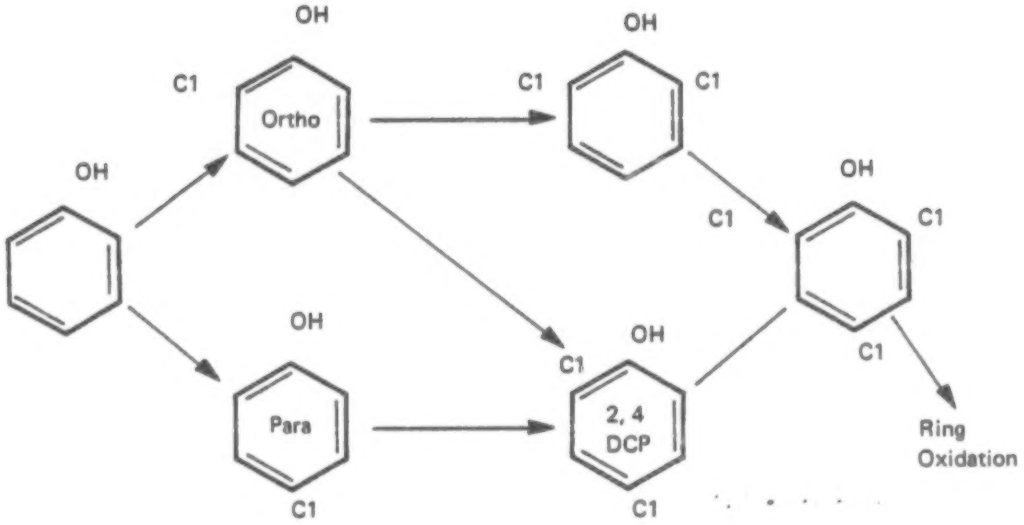
The high reactivity of phenol in the formation of chlorophenols is attributable to the ring-activating electron-releasing properties of the OH functional group (MCA 1972). The nature of the activating group is such that halogen substitution in aqueous solution is preferentially favored in the ortho- and para- positions with respect to the OH group. 2,4,6-trichlorophenol is the final step before ring oxidation. Figure 12 shows the oxidation pathways. The amount of chlorine required for the complete breakdown is greater than the amount required for the stochiometric formation of trichlorophenol. This can be attributed to the additional chlorine required for direct oxidation.

The presence of ammonia in wastewater treatment facilities inhibits the chlorination of phenol (Murphy et al. 1975, Jahnig and Bertrand 1976). The presence of inorganic chloramines may degrade phenol, as suggested by the formation of phenol oxidation products (in wastewater chlorination). In the decomposition of chloramines free residual chlorine is released which could in turn react with phenol. Therefore, decomposition of chloramines would be the rate-controlling step. In a reaction of chlorine/phenol in the presence of ammonia, the ammonia can retard the uptake of Cl through the formation of less oxidative chloramines. Given a sufficient contact time, however, phenol can be both chlorinated and oxidized, even by monochloramines with their weak potential for oxidation (Murphy et al. 1975).

Other forms of treatment for phenol are also available, and these processes have varying degrees of efficiency. Hydrogen peroxide, for example, will degrade phenol. At an initial phenol concentration of 500 mg/l, a temperature of 49°C, and initial pH of 5.5, a 4:1 ratio of H_2O_2 to phenol plus 0.01% of ferrous sulfide will lower the final phenol concentration to 3 mg in 30 minutes (Throp 1975).

Three kg of potassium permanganate per m^3 of waste containing 60-100 mg/l of phenol at a pH of 8.5-9.5 will destroy 90% of the phenol in 10 minutes. With 123 mg of phenol and about 10 mg/l of potassium permanganate, all phenol will be destroyed in 20 minutes (Throp 1975).

Ozone is used in wastewater treatment and is capable of removing phenol to almost any low-level concentration (Throp 1975). Ozone can substitute an oxygen atom onto an aromatic ring to form a phenol, but as the reaction progresses with further additions of oxygen, hydroquinones and catechol (dihydroxy compounds) will be formed (Gould and Weber 1976). These species appear at the earliest stages of ozonation, with the concentration peaking about five minutes into the reaction, then disappearing by ten minutes. There is significant removal of phenol


OH
C1
Ortho
OH
C1
C1
OH
OH
C1
C1
OH
Para
C1
OH
C1
2, 4
DCP
C1
C1
Ring
Oxidation

Phenol
0 – chlorophenol
p – chlorophenol
2,4 – dichlorophenol
2,6 – dichlorophenol
2,4,6 – trichlorophenol
Non-aromatic oxidation products

Source: Manufacturing Chemists Association, 1972.

FIGURE 12 OXIDATION PATHWAYS AND PRODUCTS FOR PHENOL

and aromatic degradation products (as indicated by a 70-80% drop in COD) after 4-6 moles of ozone per initial mole of phenol have been consumed (Gould and Weber 1976). 1.24 mg/l O_3 applied to 10 mg of phenol at 24°C will destroy 87% of the phenol in five minutes. More than 1.24 mg/l of ozone will remove all of the 110 mg of phenol (Throp 1975).

The kinetics of the ozone reaction can be expressed as (Gould and Weber 1976):

$$d[Ph]/dt = -k\,D\,[Ph]$$

where: k = a proportionality constant in the first-order rate of expression, approximately 0.132 moles phenol/mole ozone for $0 < D < 50$. The value of k is also a function of pH (see Figure 13).

D = ozone dose rate in (moles O_3/time)/(initial moles phenol)

$[Ph]$ = concentration of phenol

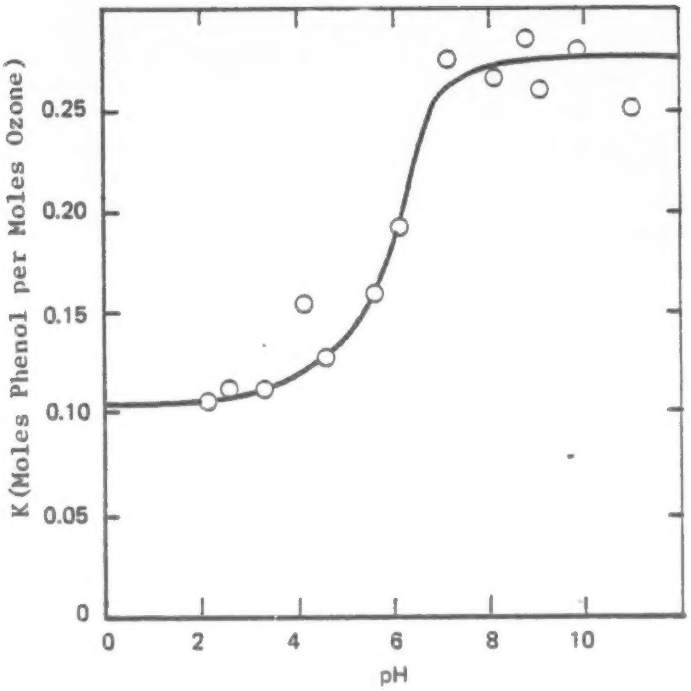
so that

$$[Ph] = [Ph]_o\, e^{-kDt}$$

Similarly, iron (VI) ferrate was found to be very effective (99%) in phenol reduction from pure water at pH 8 and a molar ratio of 15:1 (Waite and Gilbert 1978).

4. Field Studies

Limited data were available regarding the effectiveness of treatment of phenol by POTW's. In a survey of nine U.S. POTW secondary treatment plants, Burns and Roe (1980) found phenol in influent waters of all POTW's, with an average concentration of 9 ug/l and a range of 1-200 ug/l (see Table 25). The average frequency of occurrence of phenol in influents of these POTW's ranged from 33 to 100% with a mean of 78%. The removal data were highly variable. Three plants reported 99+% removal efficiencies (based on average influent versus average chlorinated effluent concentrations); a fourth achieved a 93% average. Phenol levels in sludge and solid waste following digestion were variable, ranging from 1 ug/l in floatables to 2000 ug/l in digested sludge. Due to the limited data base and period of sampling, no conclusions can be made regarding phenol accumulation in sludge. However, based on the low levels of phenol observed in sediment (Pathway #1), adsorption onto sludge would not appear to be a signficant process.



Source: Gould and Weber 1976.

FIGURE 13 RELATIONSHIP OF OZONE REACTION RATE (K) TO pH

TABLE 25. PHENOL CONCENTRATIONS IN WASTEWATER TREATMENT AT DIFFERENT STAGES OF TREATMENT (ug/l)

		Effluent	Final	%	Sludge		
Place	Influent	Pre-Clarification	Effluent	Removal	Primary	Combined	Other
Indianapolis, IN Belmont WWTP	16	5	21	-	94		68[1], 1[2]
Lewiston, ME Lewiston-Auburn WWTP	25	< 50	< 50	-	277	4,297	
Atlanta, GA R.M. Clayton WWTP	14	-	1	93		103	70[3]
St. Louis, MO Coldwater Creek STP	1	-	ND*	100	27		48[3]
Pottstown, PA Pottstown Borough STP	1	-	ND	100	882		2,000[3]
Grand Rapids, MI STP	9	< 300	-	100		173	1,717[4], 907[5]

* ND = Not Detected

Key:
1 Waste Activated Sludge
2 Floatables
3 Digested
4 Heat Treated
5 Heat Treatment Decant

Source: Burns and Roe 1980.

G. PATHWAY #5. SOIL TO GROUNDWATER AND SURFACE WATER: LEACHING, RUNOFF

1. Introduction

Phenol will sorb to some extent onto soils and organic matter and therefore may be carried away along with the soil by erosion. In soils onto which phenol does not sorb strongly, its high solubility indicates that it will leach from the soil (Versar 1979a). Phenol is present in groundwater due to leaching from strip mines and exposed coal seams and from water contact in oil and gas fields (Versar 1979a).

2. Fate Processes

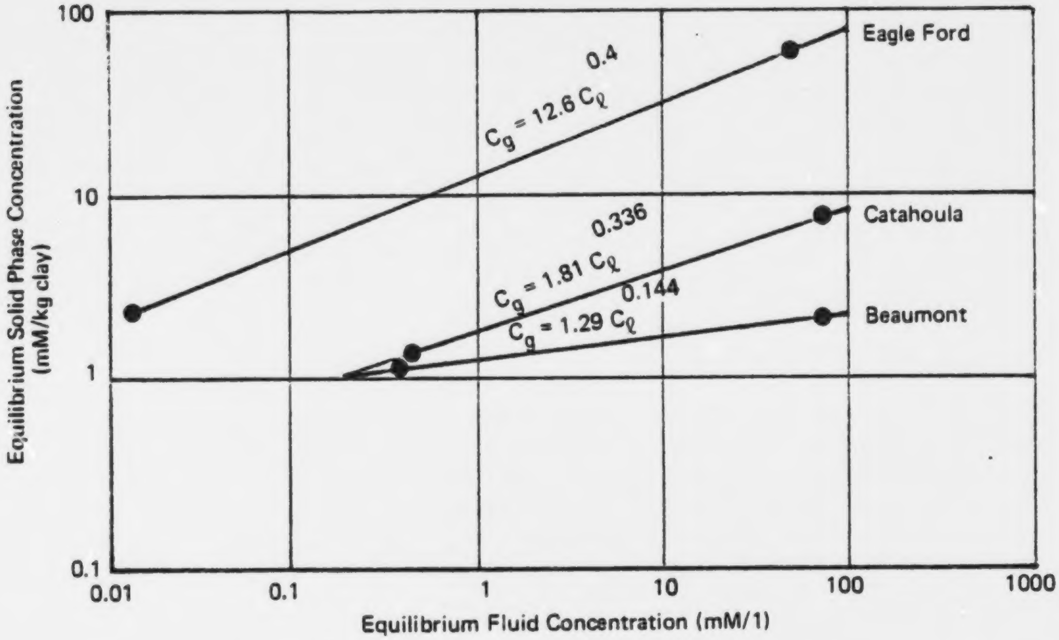
a. Adsorption

Phenol exhibits a capacity for adsorption on some soils (Sanks and Gloyna 1976, Greskovich 1974). It has a log octanol-water partition coefficient of 1.46, which indicates a low propensity for adsorption, probably onto the organic matter in soils rather than the clay fraction (Versar 1979a). In a test with three clays having the properties shown in Table 26, the isotherms for adsorption of phenol in aqueous solutions shown in Figure 14 were determined (Sanks and Gloyna 1976). Phenol was sorbed at about 1-40 x 10^{-3} mole/kg clay and showed a very low affinity for any of the clays tested. Very little or no sorption was noted on a typical Pennsylvania "claey silt" soil, as is shown in Figure 15. Phenol showed an equal affinity for both the soil and water phases (Greskovich 1974).

TABLE 26. COMPOSITION OF SOILS USED IN PHENOL ADSORPTION STUDY

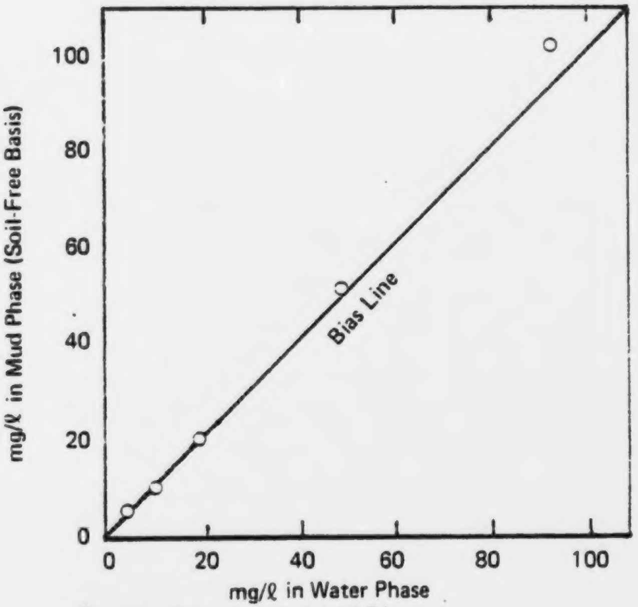
Clay Type	Beaumont	Catahoula	Eagle Ford
Grain Size, %			
Clay	58	38	47
Silt	41	54	41
Sand	1	8	12
Clay Mineral, %			
Montmorillonite	55	Most	80
Illite	45	--	20
Carbonate content, %	12	1	29
Dry Density, lb/ft^3	59	75	94
Moisture, %	30	42	26
Cation Exchange Capacity, me/100 g	53	56	20

Source: Sanks and Gloyna 1976.



Source: Sanks and Gloyna, 1976

FIGURE 14 PHENOL ADSORPTION ISOTHERMS FOR THREE TYPES OF CLAYS



Source: Greskovich 1974.

FIGURE 15 EQUILIBRIUM DATA FOR PHENOL BETWEEN THE SOIL AND WATER AT 77°F

Phenol is partially present as the phenate ion in soils. The anionic form present in soils due to reactions with metals has a λ_{max} within the absorption spectrum of natural light and thus may be subject to photolysis (Versar 1979a). Complexes of phenol with metal cations such as iron III can absorb light strongly at about 600 nm and so are also subject to photolysis (Versar 1979a). Phenol may also photooxidize on surface films (Versar 1979a); however, due to phenol's mobility in soil, only a small amount would likely be subject to either process and for a short duration.

b. Volatilization

Phenol may volatilize slowly from soils (Versar 1979a), but no rate constant is available. A method will be discussed here from which a rate constant can be computed. The process of volatilization from soils is much more difficult to describe than volatilization from water, as it is dependent on many factors; soil water content, soil type, soil bulk density, soil organic-matter content, sorption properties, depth to which the chemical is incorporated into the soil, wind speed, temperature, humidity, diffusion coefficient of the chemical, and other chemical properties such as vapor pressure and solubility. Since phenol apparently has a low capacity for sorption and has a rather high solubility, it will likely be found in the soil water rather than sorbed onto soil particles when soil water is present. In very dry soils, however, the low vapor pressure of phenol would tend to cause it to diffuse slowly in the vapor phase. It would therefore remain loosely associated with soil particles until water reaches the sorption sites and is preferentially sorbed, thus displacing the phenol.

No accurate methods incorporating all the factors mentioned above exist for determining a volatilization rate from soil. Some models have been developed, but do not address the complexities of environmental conditions. Dow (Swann *et al*. 1979) has tested nine pesticides for volatilization from soil surfaces and has presented a prediction method which agrees fairly closely with measurements of their volatilization rates. The rate constant was proportional to P_{vp}/SK_{oc}, where P_{vp} is the vapor pressure (0.5293 mm Hg for phenol), S is solubility (93000 mg/l for phenol), and K_{oc} is the soil adsorption constant (4.3, assumed equal to K_{ow}), so that $P_{vp}/SK_{oc} = 1.32 \times 10^{-6}$. This number is at the top of the range of values computed for the pesticides and corresponds to a predicted half-life at 0.24 hours ($t_{1/2} = 3.8 \times 10^{-7} SK_{oc}/P_{vp}$ hours) and a rate constant of 2.4/hr. This is rapid relative to the other pesticides tested.

Phenol was almost completely lost from montmorillonite clay exposed for one week to an atmosphere of 40% relative humidity (Versar 1979a). This observation supports the estimation of a short half-life described previously. Other processes, such as photodegradation, may have also attributed to the observed loss.

The vapor pressure and solubility of phenol are considerably higher than those of most pesticides, and the sorption coefficient is much lower. This may affect the correlation based on pesticide data. Caution is urged in using this rate since there is little evidence to verify it.

c. Other Processes

Three other fate processes may affect phenol concentrations in soil: biodegradation, chemical oxidation, and complexation. No specific information on phenol biodegradation in soil was available; however, due to the susceptibility of the compound in aquatic systems (see Pathway #1) by biological oxidation, it is assumed that under aerobic conditions this reaction would also be supported in soil. In aerobic soils containing metals, non-photolytic chemical oxidation of phenol may also occur (Versar 1979a), although no specific information was available. It is also probable that phenol forms complexes with metal complexes (e.g., ferric iron) in soil; however, again no specific information was found investigating this phenomenon (Versar 1979a).

Bioaccumulation of phenol in terrestrial plants from soil or irrigation water is not expected due to its short persistence time and low octanol-water partition coefficient (Versar 1979b). Spinach was found to absorb less than 1% of phenol applied in solution at concentrations of 2 mg/l to 200 mg/l (Mueller 1975).

3. Field Studies

There is a potential for certain soils to retain phenol and prevent movement into groundwater. In a study in Upper Vistula Floodplain, Poland (Kleczkowski *et al*. 1972), soil was monitored in the vicinity of a catchpit (sewage sedimentation pit) of a chemical plant in which phenol was stored with other organics. The surrounding substratum was composed of peat, sand, and impervious silt. One hundred and twenty analyses from 44 locations (with piezometers and village wells) found the range of phenol pollution limited to 500 meters from source. This was attributed to attenuation by the peat. No information was given on the concentrations present.

Another study by Delfino and Dube (Delfino and Dube 1976) was of an accidental phenol spill in Wisconsin. The substance spilled was carbolic acid (95-100% phenol). The incident, which occurred in June, was caused by a railroad tanker derailment and the total spill amounted to 35,000 liters (out of a total of 80,000 liters). Residues of the incident persisted for 19 months making well water in the area nonpotable. Sand and gravel aquifers and undifferentiated dolomite were characteristic of the substratum in the area which resulted in slow groundwater movement through shallow flow paths and discharge to small streams. The chemical form of the phenol was liquid (melting point, 41°C) due to the summer heat and the residual heat in the chemical itself.

Some of the spill was recovered in solid form following cooling; the rest percolated into the soil and was mobilized by precipitation and runoff moving into the aquifer. Well depths in the area were 23-30 meters. Phenol was monitored as far away as 390 meters. Movement was primarily to the southeast. Control wells beyond the path showed "phenol-like" concentrations of 0.001-0.10 mg/l which were natural phenolics. There were no chlorinated water supplies in the vicinity of the spill so no chlorophenols formed. The authors of the study suspected this would have occurred had chlorine been used as part of local water treatment practices.

4. Estimation Methods

The EPA Office of Solid Waste (U.S. EPA 1980c) evaluated the potential for and rate of phenol movement from generalized unconfined landfills and lagoons into surface water for two waste streams. In both cases, all phenol present was assumed to be mobilized. Table 27 presents the estimated release rates.

TABLE 27. ESTIMATED RELEASE RATES OF PHENOL IN SOLID WASTE FROM WASTE DISPOSAL SITES

	Annual Release Rate to Surface Water	
	Landfills (kg/m^2)	Lagoons (kg/m^2)
Waste Stream #1 [1]	10 - 40	148
Waste Stream #2	19 - 76	280

---

[1] Waste streams not identified.

Source: U.S. EPA 1980 c.

Two known dischargers of phenol-containing solid waste are producers using cumene peroxidation and benzene sulfonation (see Section III). Assuming the discharge rates estimated in Appendix A No. 24, the two largest producers using these landfill or lagoon methods would annually create 990 kg and 1,260 kg of solid waste, respectively. Assuming a land disposal site of 30 to 125 m^2 and 100% mobilization of all phenol present, the release rate of Waste Stream #1 for landfill could easily be achieved. A loading rate of phenol to nearby surface water can be estimated for the benzene sulfonation process assuming: 1) all of the waste is deposited at the same location and runoff drains into the same water body, and 2) as a conservative estimate, all phenol present migrates in one half of a year. The estimated loading rate is approximately 7 kg/day or, distributing the loading over 24 hours per day, 0.3 kg/hr. This rate is low compared to other estimated rates (see Tables 20 and 21). Based on this estimation, the solid waste of phenol, even if 100% mobile, contributes a lower localized aquatic discharge rate of phenol than does direct discharge in effluents. However, an industry producing four times the amount of solid waste produced by the benzene sulfonation plant (∿5,000 kg, benzene sulfonation plant), would have a loading rate more comparable to some of the direct aquatic waste dischargers (∿1.5 kg/hr). Information on phenol solid waste generation is incomplete and does not cover all industries so it is not possible to assess whether a significant solid waste discharger exists.

5. Monitoring Data

No monitoring data were available measuring phenol concentrations in soil and in groundwater for both natural background levels and in relation to intentional releases.

H. PATHWAY #6. CHLORINATION OF PHENOL AND FORMATION OF CHLOROPHENOL

Phenol is one of the most reactive of the aromatics under conditions of dilute aqueous chlorination (Carlson and Caple 1975, Carlson *et al*. 1976). Table 28 presents data from a chlorination experiment with pH as a variable.

TABLE 28. CHLORINE[1] INCORPORATION IN PHENOL[2]

	% Chlorine remaining after reaction time[3]
pH 3	2.2 ± 0.1
pH 7	2.4 ± 0.1
pH 10	2.4 ± 0.1

---

[1] Chlorine at 7.0×10^{-4}M,

[2] Phenol at $9.5 \pm 0.6 \times 10^{-4}$M

[3] 20 minutes at 25°C.

Source: Adapted from Carlson and Caple 1975.

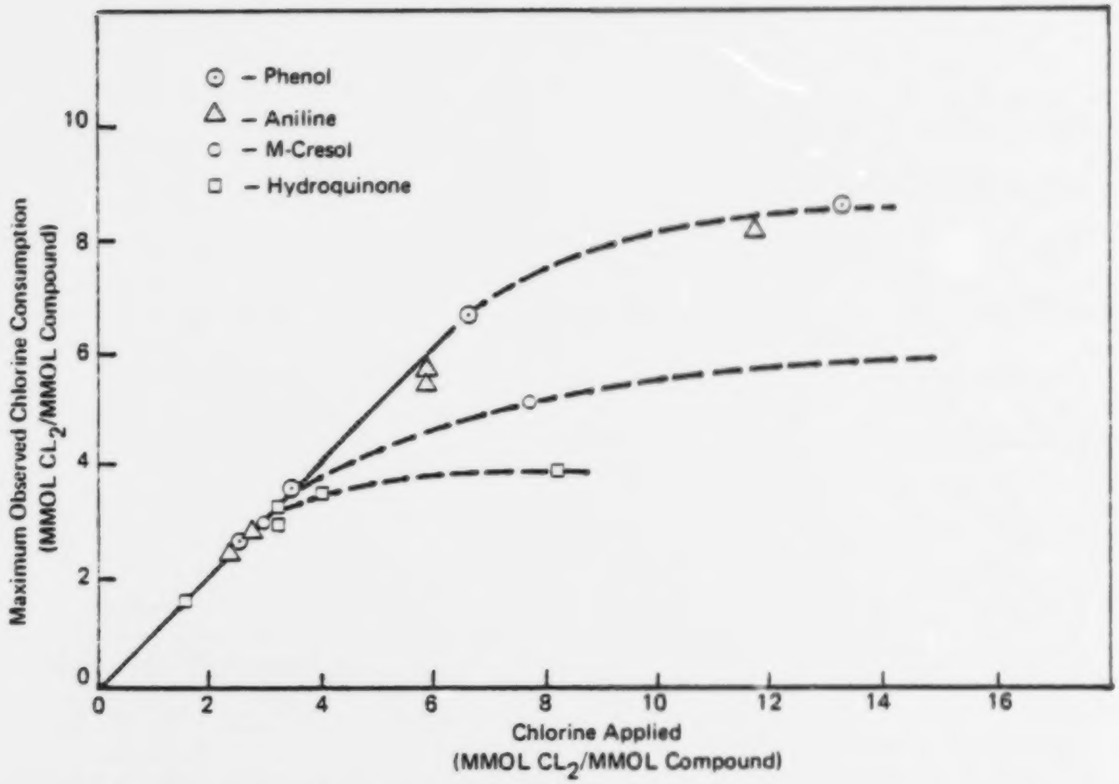
Chlorination was independent of pH, indicating the likelihood of chlorination in a wide range of treatment processes using chlorine.

When sufficient chlorine is supplied and enough contact time is allowed (1 hr), the net chlorine demand by phenols is approximately 8 mmol Cl_2 for each mmol of phenol, or 6.35 mg Cl_2/mg phenol. Table 29 and Figure 16 present the results of an experiment measuring this demand (MCA 1972).

TABLE 29. CHLORINATION OF PHENOL

Phenol Concentration (mg/l)	Applied Chlorine (mg/l)	Contact Time (hr)	Chlorine Residual (mg/l)	Net Chlorine Demand	
				(mg/l)	$\frac{(\text{mmol } Cl_2)}{\text{mmol phenol}}$
10	20	0.25	6.0	14	1.9
		0.5	1.7	18.3	2.4
		1.0	0	≥20	≥2.7
		2.0	0	≥20	≥2.7
10	50	0.25	17.8	32.2	4.3
		0.5	8.0	42	5.6
		1.0	2.8	47.2	6.3
		2.0	0	≥50	6.6
10	100	0.25	50	50	6.6
		0.5	44	56	7.4
		1.0	39	61	8.1
		2.0	36.5	63.5	8.4
20	50	0.25	4.5	45.5	3.0
		0.5	0	≥50	≥3.3
		1.0	0	≥50	≥3.3
		2.0	0	≥50	≥3.3
20	100	0.25	12	88	5.8
		0.5	2.5	97.5	6.5
		1.0	0	≥100	≥6.6
		2.0	0	≥100	≥6.6

Source: MCA 1972.



Source: Manuafacturing Chemists Association 1972.

FIGURE 16 MOLAR CHLORINE UPTAKE BY TEST COMPOUNDS, INCLUDING PHENOL

The most commonly formed products of phenol chlorination are chlorophenol (o- and p-), 2,4-dichlorophenol, 2,6-dichlorophenol, and 2,4,6-trichlorophenol (MCA 1972).

Chlorination of phenols can occur during drinking water finishing, wastewater treatment, and in cooling towers. The concentration of chlorine available for reaction ranges from 1-50 mg/l, depending on the the type of treatment (see Table 30). Although not all the chlorine present in actual systems will react with phenol due to competition from other compounds (e.g., benzene, biphenyls), for the purpose of estimation it is assumed 100% available to phenol based on the results described in Table 30. In one liter of water in various types of water treatment, 0.25 to 10 mg of chlorine are present. Assuming the optimum chlorine demand measured in laboratory studies (Table 29 and Figure 16), then approximately 6.35 mg of Cl_2 are consumed by each mg of phenol. Therefore 0.0 to 1.6 mg of phenol would be chlorinated in each liter of water during drinking water or typical wastewater treatment in one hour. This value provides an upper limit on the concentration of phenol that can undergo chlorination. However, due to variabilities in wastewater treatment, typically shorter wastewater retention times during treatment, and the likelihood of formation of more chlorinated phenolic compounds, the actual concentration of phenol chlorinated during treatment is lower.

TABLE 30. CHLORINE LEVELS IN WATER TREATMENT

	mg/l
Drinking Water Treatment	1.0 - 16.0
Groundwater Wells	0.25 - 7.0
Main Sterilization (in reservoirs, ship tanks)	50[1]
Wastewater Treatment (secondary effluent)	10 - 15
Cooling Towers	1 - 2

---

[1] With retention time of 24 hours.

Source: White 1975.

Due to the propensity for reaction of phenol with chlorine, it would be expected that: 1) high phenol levels in wastewater plant influents would be greatly reduced in the effluent, and 2) chlorinated phenol levels in the effluent would exceed influent levels. Unfortunately, The most commonly formed products of phenol chlorination are chlorophenol (o- and p-), 2,4-dichlorophenol, 2,6-dichlorophenol, and 2,4,6-trichlorophenol (MCA 1972).

Chlorination of phenols can occur during drinking water finishing, wastewater treatment, and in cooling towers. The concentration of chlorine available for reaction ranges from 1-50 mg/l, depending on the the type of treatment (see Table 30). Although not all the chlorine present in actual systems will react with phenol due to competition from other compounds (e.g., benzene, biphenyls), for the purpose of estimation it is assumed 100% available to phenol based on the results described in Table 30. In one liter of water in various types of water treatment, 0.25 to 10 mg of chlorine are present. Assuming the optimum chlorine demand measured in laboratory studies (Table 29 and Figure 16), then approximately 6.35 mg of Cl_2 are consumed by each mg of phenol. Therefore 0.0 to 1.6 mg of phenol would be chlorinated in each liter of water during drinking water or typical wastewater treatment in one hour. This value provides an upper limit on the concentration of phenol that can undergo chlorination. However, due to variabilities in wastewater treatment, typically shorter wastewater retention times during treatment, and the likelihood of formation of more chlorinated phenolic compounds, the actual concentration of phenol chlorinated during treatment is lower.

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---

[1] With retention time of 24 hours.

Source: White 1975.

Due to the propensity for reaction of phenol with chlorine, it would be expected that: 1) high phenol levels in wastewater plant influents would be greatly reduced in the effluent, and 2) chlorinated phenol levels in the effluent would exceed influent levels. Unfortunately, little data are available on phenol concentrations at various stages of wastewater treatment so that the mass of phenol removed by microbial degradation and other processes can be subtracted from the amount available for final chlorination. Additionally the same slug of phenol is seldom followed throughout the entire treatment process so that the effluent level reported may be for a larger loading than the level indicated in the influent. In Pathway #3 the behavior of phenol in wastewater treatment was described and close to 100% efficiencies in removal were reported (Burns and Roe 1980). Since phenol itself (and not phenols) was sampled it is possible that some of the amount "removed" was actually chlorinated and present in the effluent as chlorophenols. If the data for chlorophenols from the same sampling program is examined, it can be seen that sometimes effluent levels exceed influent levels but again the data are erratic. Table 31 presents some concentrations of phenol and chlorinated phenol reported in sewage treatment influents and effluents.

TABLE 31. PHENOL CONCENTRATIONS IN POTW'S

Chemical	Influent Levels (ug/l)	Effluent Levels (ug/l)	Reference
Phenol	1 - 25 (6 plants)	0 - <50	Burns and Roe 1980
2,4,6-trichlorophenol	0 (2 plants)	<2 - <50	Burns and Roe 1980
2,4 dichlorophenol	<1 - 1 (2 plants)	<1	Burns and Roe 1980
2-chlorophenol	<50 (1 plants)	<50	Burns and Roe 1980
Chlorinated phenols	not given	∿0.5 - 1.5	Jolley et al. 1975

Phenol has rarely been detected in drinking water, both raw and finished (see Section V-B). When detected, levels have been very small, on the order of 1-5 ug/l.

In conclusion, chlorination of phenol not subject to degradation appears likely during wastewater treatment. Phenol discharged to POTW's is first likely to be biodegraded or chemically treated (as described in Pathway #3) before contact with chlorine. Some phenol reaching the chlorination stage would presumably be chlorinated at the chlorine levels commonly maintained. Few monitoring data are available to confirm this hypothesis, especially on phenol concentrations between secondary treatment and chlorination. The limited data suggest removal of phenol during treatment (whether due to degradation or conversion to chlorinated products). Some data for chlorophenols indicate formation of chlorophenol during treatment; other data are not conclusive.

A separate risk assessment on chlorinated phenols (chlorophenol; 2,4-dichlorophenol; and 2,4,6-trichlorophenol) (Scow et al. 1980) considers the production and use, environmental fate and distribution, effects and exposure, and risk in regard to these compounds.

I. SUMMARY AND CONCLUSIONS

Following release to the environment, the fate and distribution of phenol depends on the form of emission, the receiving medium, and various environmental factors. Five environmental pathways describing the behavior of phenol releases were identified: emissions to surface water; emissions to air; transport from air to water/soil; discharges to POTW's; and releases to soil. A sixth pathway, chlorination during water treatment, was also considered.

1. Surface Water

Approximately 34% of all known environmental releases of phenol are made to surface water, primarily by POTW's, bisphenol A producers, petroleum refiners, and certain producers of phenol itself. The most significant fate process affecting phenol in surface water is biodegradation. A half-life of 3.5 days was reported under field conditions in a river. Laboratory studies confirm a rapid removal rate especially under acclimated conditions and at high temperatures. Numerous microfloral species have been identified as capable of degrading phenol. There is some evidence that phenol may undergo photolysis under environmental conditions. The processes of hydrolysis, oxidation, adsorption, and volatilization do not appear to be significant with respect to phenol concentrations in surface water.

Studies of the bioaccumulation of phenol have been conducted on aquatic organisms. Absorption is the primary route of intake. Phenol concentrations of 14 to 156 ug/gram of body weight were reported for goldfish exposed to 10-100 mg/l phenol for 1-5 days. The bioconcentration factors of phenol were low, ranging from 1.2 to 2.3 above the water levels. Phenol tends to accumulate in greater amounts in the gall bladder, liver, and visceral organs. Higher vertebrates (e.g., mammals) have the ability to detoxify phenol by forming conjugation products with glucuronides and sulfates; fish do not appear to possess this mechanism, rather, passive diffusion and biliary excretion are the mechanisms of decreasing the phenol body burden. No direct evidence was obtained for biomagnification of phenol; however, it does not appear to be significant because of its generally low degree of accumulation in tissue.

In a field study of a monohydric phenols spill from a benzene sulfonation plant into a river, 93% of the initial 28 mg/l phenol concentration was reduced in six days. Associated with the high rate of phenol degradation was a temporary deficit in oxygen levels which contributed to the toxic effects on the system resulting from the spill. Phenol concentrations may be higher during the winter than summer due to a decreased rate of microbial degradation at low temperatures.

The EXAMS model simulation of a continual 3 kg/hr discharge of phenol into a eutrophic lake and turbid river (1 km length) estimated equilibrium water column concentrations of 1 ug/l to 3 ug/l. The self-purification time for both systems was approximately 3 to 4 hours due to biodegradation in the eutrophic lake and physical transport in the river system. Sediment concentrations were approximately 10 ug/kg and 5×10^{-4} ug/kg (dry weight) in the river and lake systems, respectively.

Monitoring data for phenol in surface water are limited. The STORET data base reports a total of approximately 600 observations for thirteen major river basins. Ninety-three percent of all observations were remarked data, i.e., either at or below the detection limit (∿10-50 ug/l). Mean concentrations for unremarked data between 1978 and 1980 ranged from 0.004 ug/l to 660 ug/l with a maximum of 6,794 ug/l. Mean sediment levels (38 observations) averaged 102 mg/kg (unremarked data) with a maximum value of 454 mg/kg. Concentrations as high as 3,000 mg/l have been reported in effluents; however, most levels were reported at less than 10 mg/l.

2. Emissions to Air

Approximately 59% of the estimated environmental releases of phenol are to air, predominately from combustion of wood. Other releases are from phenol producers, consumers and from transport and storage of phenol. Most releases are presumably in vapor form, some of which is sorbed onto particulate matter. Phenol is subject either to rainout or to photolysis and photooxidation with an estimated atmospheric lifetime of several days. Phenol levles in urban areas fluctuate on a daily basis with higher levels during the day, apparently due to higher traffic volume and industrial activity. The highest reported phenol concentration in urban air was 289 ug/m^3. No data were available for rural areas.

3. Rainout

The amount of atmospheric emission of phenol not photodegraded is subject to rainout and transfer to land or surface water. Initial concentrations in rainwater were estimated at 1 to 10 mg/l in the vicinity of a source; however the concentrations would be continually reduced as rainfall continued. No monitoring data were available reporting phenol levels in rain; however industrial areas are reported to have qualitatively higher rainfall concentrations than rural areas.

4. Fate in POTW's and Wastewater Treatment

Phenol is discharged to POTW's both in untreated and treated waste streams by various industrial sources. In addition, natural background levels of phenol contribute some portion of the total loading. Typical POTW influent concentrations measure 1 ug/l to 200 ug/l. Numerous treatment processes are effective in removing phenol by degradation including activated sludge, trickling filters and various chemical treatments. Biodegradation in sludge is very effective, especially in activated sludge at concentrations less than 10 mg/l. Above this level, the decay rate is sometimes inhibited. At about 500 mg/l, one activated sludge system experienced a sharp disruption of microbial activity. The optimum pH range is 6 to 9.5 and adequate essential nutrient concentrations must be present.

Chemical removal methods effective in degrading phenol include treatment with chlorine, hydrogen peroxide, potassium permanganate, ozone, and iron ferrate. The prevalence of these methods is expected to be small in POTW's and variable in wastewater treatment facilities depending on the industry subcategory.

A field study investigating the fate of phenol during treatment in seven POTW's reported a high removal efficiency of greater than 90% for four plants. Efficiencies were not reported for the other three plants.

5. Soil to Groundwater and Surface Water

Only a small percentage (approximately 7%) of the total environmental releases of phenol are made to land. Major releases are made in the disposal of sludges resulting from the synthesis of phenol. Due to phenol's relatively low affinity for adsorption onto soil and its high solubility, some portion of the land releases is expected to reach either ground or surface waters unless significantly reduced by biodegradation or other fate processes.

The most important fate process determining phenol concentrations in biologically active soil is expected to be biodegradation based on its effectiveness in degrading phenol in aquatic systems. Volatilization from soil does not appear to be significant. No information specifically regarding chemical oxidation and complexing of phenol was available.

Two field studies investigating the behavior of phenol in soil indicated attenuation and persistence of the chemical in the vicinity of the source. In a peat soil, the range of phenol was limited to within 500 m of a catchment pit indicating in contradiction of laboratory results, strong adsorption onto organic matter. In the second study phenol spilled onto the soil surface reached groundwater supplies and persisted for 19 months. The substratum in the vicinity of the spill was sand, gravel, and undifferentiated dolomite. There was no indication of adsorption onto the organic fraction of the soil surface layer; however, soil concentrations were not reported to confirm this.

6. Chlorination of Phenol and Formation of Chlorophenols

Phenol is one of the most reactive of the aromatics in regard to chlorination. Formation of chlorophenols during treatment of wastewater and drinking water is commonly reported. Based on the results of an experiment conducted under optimum conditions, an upper limit of 1.6 mg of phenol per liter of water will undergo chlorination at typical wastewater treatment chlorine concentrations. Lower concentrations, however, are actually expected to be subject to reaction with chlorine due to system variability and stereochemical factors. Field studies are too few and inconsistent to support any specific conclusions about the propensity of chlorination of phenol during POTW treatment of wastewater.

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SECTION V.

HUMAN EFFECTS AND EXPOSURE

A. EFFECTS ON HUMANS

1. Introduction

Phenol was first used as a medicinal disinfectant more than a century ago and, although largely replaced by other compounds, it is still found in throat lozenges, mouthwashes, and other medicinal preparations. Humans also produce phenol endogenously via bacterial action on ℓ-tyrosine in the intestinal tract. This section of the report will examine the adverse health effects associated with phenol exposure.

2. Metabolism and Bioaccumulation

Phenol is readily absorbed from all routes of entry followed by rapid generalized distribution in the body and relatively rapid metabolism and excretion. In man, inhalation exposure to phenol vapor (via face mask) resulted in an initial lung retention of 80% of the dose. This value dropped with time to an average of 70% after 6 to 8 hours (Piotrowski 1971).

In a similar experiment in which human volunteers were exposed to phenol vapor in a chamber but allowed to breathe fresh air through a face mask, Piotrowski(1971) found phenol vapor was readily absorbed through intact skin.

Pullin also reported rapid dermal absorption of phenol (0.05% of body weight) applied over 35-40% of the total body surface area of swine for one minute followed by 15 minutes of showering with water. Peak blood concentrations of phenol occurred 15 to 30 minutes after application (Pullin *et al*. 1976).

Phenol's moderate water solubility and lipid/water partition coefficient favor facile transfer through membranes and biological barriers. Deichmann reported that within 15 minutes following oral administration of 500 mg of phenol/kg in the diet to rabbits highest tissue concentrations were found in liver (637 mg free phenol/kg tissue) followed by heart (530), lungs (342), kidney (353), blood (308), and muscle (190). Phenol concentrations became fairly uniform as time progressed and began to decrease as metabolism occurred (Deichmann 1944).

A similar distribution scheme was noted in the desert rodent, *Notomys alexis*, following intraperitoneal injection of 5, 25, or 100 mg/kg phenol (Wheldrake *et al*. 1977). Kao reported 84-90% urinary elimination of 25 mg/kg ^{14}C-phenol within 8 hours in sheep, pigs, and rats (Kao *et al*. 1979). Phenyl glucuronide accounted for 49, 83, and 42%, respectively, of the urinary metabolites, while sulfate conjugates accounted for an additional 32, 1, and 55%, respectively, of the urinary metabolites in these species. Conjugates of quinol were minor urinary metabolites (<7%) in all three species. Less than 0.5% of the dose was excreted in feces, indicating almost complete absorption of phenol had occurred.

Once absorbed and distributed in the body, phenol undergoes two main metabolic reactions: (1) conjugation of the hydroxyl group with glucuronic acid and/or (2) conjugation with sulfuric acid to form ethereal sulfates. Some species differences are seen with respect to minor reactions. Hydroxylation to form quinol in rabbits, rats, sheep, and pigs (Williams 1959, Kao _et al_. 1979) and methylation of the phenolic hydroxyl group in rabbits (Williams 1959) have been reported. In the cat, up to 8% of the dose was excreted as phenyl dihydrogen phosphate (Capel _et al_. 1974), while some 12% of the urinary metabolites in sheep were conjugated with phosphate (Kao _et al_. 1979). In man, the sulfate conjugates appear to predominate (Capel _et al_. 1972).

Glucuronic acid conjugation of phenol generally exceeds that of sulfate conjugation when the dose of phenol is relatively large. This appears to be due to the fact that the rate of glucuronic acid conjugation is proportional to the body level of phenol, whereas the rate of sulfate conjugation is independent of the phenol level but dependent on the availability of sulfate (Williams 1959). For example, the ratio of sulfate to glucuronide metabolites in the desert rodent, _Notomys alexis_, decreased with increasing dose of phenol, while the level of glucuronides showed a contrasting increase with dose. Phenyl sulfate predominated following intraperitoneal injection of 5 mg/kg phenol (57% vs. 26% glucuronide) but its proportion decreased as the level of injected phenol increased (i.e., at 25 and 100 mg/kg, the ratios of sulfate to glucuronide were 27:32 and 17:36, respectively (Wheldrake _et al._ 1978). Similar dose-related excretion patterns have been found in several species of primates (Mehta _et al_. 1978).

In mammals, phenol is produced endogenously by the degradative action of bacteria on tyrosine in the gut. Thus, a large number of phenolic substances occur both free and in conjugated form in normal urine (Williams 1959). Between 1.5 and 5 mg of phenol are normally excreted per liter of human urine per day (Fishbeck _et al_. 1975). Phenol levels in normal blood as determined by gas-liquid chromatography range from 0.04 to 0.56 mg/l free phenol plus 1.06 to 5.18 mg/l conjugated phenols (Dirmikis and Darbre 1974) and 2-18 mg/l for total phenol (VanHaaften and Sie 1965).

The clearance of exogenous phenol from the body is relatively rapid. The half-life of phenol in man determined after inhalation or skin absorption is approximately 3.5 hours (Piotrowski 1971). There are no data to suggest that bioaccumulation occurs.

3. Animal Studies

a. Carcinogenicity

In one study at NCI, addition of 0.25% or 0.5% phenol by volume for 103 weeks to the drinking water of male and female F344 rats and B6C3F1 mice resulted in an increased incidence of pheochromocytomas and leukemia or lymphomas in low-dose male rats. This may have been associated with phenol administration. However, the incidence of these tumors in high-dose male rats was comparable to control values. Thus, an association with the administration of phenol was not clearly established. Under these test conditions, phenol was not considered carcinogenic for either male or female F344 rats or male and female B6C3F1 mice (NCI 1980). However, the Data Evaluation/Risk Asssessment subgroup of NCI's Clearinghouse on Environmental Carcinogens has recommended that phenol be tested further to clarify the finding of elevated incidences of the above-named tumors (J. Sontag, Assistant Director for Interagency Affairs, NCI, personal communication, 1980).

The capacity of phenol to elicit epithelial tumors in mice exposed to a single application of a known carcinogen (i.e., initiation) followed by repeated skin painting of phenol to the same area (i.e., promotion) has been demonstrated by a number of investigators (Boutwell and Bosch 1959, Salaman and Glendenning 1957).

Benign tumors developed rapidly and in significant numbers in mice specially inbred for sensitivity to tumor development following a single skin application of 75 ug of 9, 10-dimethyl-1,2-benzanthracene (DMBA) followed one week later by repetitive twice-weekly applications of 2.5 mg phenol (as a 10% solution in benzene) to the same area for 42 weeks. After 13 weeks, 22 of 23 mice (96%) had papillomas and 73% had developed carcinomas. Few tumors developed on mice treated either with DMBA alone (3/21 mice with papillomas at 42 weeks) or with phenol alone (5/14 papillomas at one year). Continuation of skin painting with phenol alone for an additional 20 weeks resulted in one fibrosarcoma. A few papillomas were seen in other strains of mice (Holtzman, CAF_1 C3H) similarly treated, but at much lower incidences (Boutwell and Bosch 1959).

Salaman and Glendenning reported similar results in "S"-strain albino mice after initiation with 0.3 mg DMBA followed by repeated skin application of phenol (Salaman and Glendenning 1957). A twenty percent solution of phenol plus DMBA induced marked skin trauma and a tumor incidence of 85% in 13 survivors at 37 weeks (10 weeks after treatment ceased). Phenol alone at this concentration was mildly tumorigenic, i.e., 39% tumor incidence (all benign) at 45 weeks. DMBA plus a five percent phenol solution resulted in a tumor incidence of 28% in 14 survivors at 37 weeks. Two of a total of nine tumors were malignant. No tumors were seen in mice treated with five percent phenol alone for 32 weeks.

As a result of these findings, a comprehensive study of the effects of dose and purity of phenol was undertaken. The tumor response of groups of mice exposed to graded amounts of phenol following a single application of DMBA showed that a maximal response was reached at the level of 2.5 mg phenol/mouse (10% solution) twice a week. A lesser response was obtained at 1.25 mg/mouse while 5.0 mg/mouse (20% solution) caused a number of deaths as a result of systemic toxicity. In addition, the development of papillomas was partially inhibited by the corrosive effects on the skin at the 5 mg/mouse level (Boutwell and Bosch 1959).

Since coal tar is a common source of phenol, the possibility of contamination of phenol with carcinogens was considered. Careful laboratory purification and subsequent treatment, however, resulted in no loss of carcinogenic activity (Boutwell and Bosch 1959).

In a series of studies, Van Duuren and others (Van Duuren et al. 1971, 1973; Van Duuren and Goldschmidt 1976) examined the potential cocarcinogenic activity of phenol with benzo(a)pyrene (BaP) in mice. Cocarcinogenesis is distinguished from initiation/promotion in that two or more agents are applied simultaneously or alternatively in single or repeated doses. No cocarcinogenic activity was found when reagent-grade phenol (3 mg/mouse, 3 times/week for 52 weeks) was applied simultaneously with 5 ug BaP to the backs of ICR/Ha Swiss mice. Seven of 39 survivors (18%) at 52 weeks had tumors compared to 13/42 mice (31%) receiving BaP alone and 0% in the solvent control group (Van Duuren et al. 1973). In addition, a slight inhibitory effect on tumor formation was noted, i.e., it took 267 days for the development of the first papilloma in the phenol-BaP group in contrast to 251 days in mice treated with BaP alone (Van Duuren and Goldschmidt 1976).

In summary, the addition of 0.5% phenol to drinking water was not found to be carcinogenic to either rats or mice. Phenol does appear to have tumor-promoting activity in mice but no cocarcinogenic action. Skin application of phenol alone does result in carcinogenic activity in sensitive strains of mice but not in standard inbred mouse strains. This tumorigenic activity appears to be associated with phenol's irritancy and subsequent skin hyperplasia.

b. Mutagenicity

Several bacterial mutagenicity assays have been conducted with phenol. Demerec reported that phenol produced back-mutations in Escherichia coli from streptomycin-resistance to streptomycin-sensitivity, but only at concentrations that were toxic to the bacterium (survival was only 0.5 to 1.7% at phenol concentrations of 0.2 to 0.1%) (Demerec et al. 1951). In another study, Dickey and others found phenol was not mutagenic in Neurospora (Dickey et al. 1949). More recently, Cotruvo reported phenol was not mutagenic in the bacterium Salmonella typhimurium or in the yeast Saccharomyces cerevisiae D3 (Cotruvo et al. 1977).

In *Drosophila* (fruit fly), exposure of explanted and subsequently reimplanted larval ovary to phenol (0.01%) for 15-20 minutes produced an increased frequency of lethal mutations (11.3%) compared to 0% in similarly treated controls (Hadorn and Niggli 1946).

More recently, Bulsiewicz examined the influence of phenol on chromosomes in the process of spermatogenesis in Porton-strain mice for five consecutive generations (Bulsiewicz 1977). Mice in each generation were given two milliliters of 0, 0.08, 0.8, or 8 mg/1 aqueous solutions of phenol daily by gavage for 30 days or approximately 0, 6.4, 64, and 640 ug phenol/kg/day, respectively. Six males and females from each group per generation were then mated; the females continued to receive phenol during pregnancy and lactation. Testes from six males per group for each generation were examined for chromosomal defects in spermatogonia and primary spermatocytes. Aberrations noted included chromatid and chromosome breaks, ring chromosomes, centric fusions, acentric fragments, aneuploidy, and polyploidy. A smaller number of total aberrations was seen in primary spermatocytes (5, 22, and 24% in the 6.4, 64, and 640 ug/kg groups, respectively) in comparison to the higher numbers noted in spermatogonia (27, 52, and 81%, respectively). The reasons for this difference are difficult to pinpoint. Three possible explanations are: (1) gross abnormalities in spermatogonia may have been eliminated at that stage of spermatogenesis, (2) some aberrations were corrected by normal repair processes, and (3) the responses in spermatogonia and spermatocytes are independent of each other, with the higher incidence in spermatogonia reflecting the rapidly dividing nature of these cells.

As can be seen in Tables 32 and 33, dose-related increases in the incidence of aberrations were found in both spermatogonia and spermatocytes. The highest dose, however, was associated with systemic toxicity and mortality. Apart from dose-related increases in aberrations, an apparent trend toward increased aberrations in each successive generation was noted. However, the experimental protocol as well as the inadequacy of information presented in the paper make interpretation of this latter point difficult. The issue is complicated by the facts that (1) both females and males were exposed to phenol prior to mating, and (2) both sexes of the F_1 through F_5 generations were additionally exposed to phenol *in utero*. The significance of increased aberrations in subsequent generations within a single treatment group is difficult to assess in that one cannot segregate which of three insults (i.e., to parental male, to parental female, and to offspring *in utero*) or combinations thereof contributed to the incidence of reported aberrations. Furthermore, although sterility does not appear to have been a problem, the effect of phenol exposure on other indices of fertility in progeny could not be assessed because no data on reproductive parameters or effects of exposure in females were provided.

TABLE 32. INCIDENCE OF CHROMOSOMAL ABERRATIONS IN SPERMATOGONIA OF PHENOL-TREATED MICE

Generation	Dosage Level (ug/kg/day)	% Chromosome Breaks	% Chromatid Breaks	% Aneuploidy	% Polyploidy	% Associations
P	0	0	0.8	0	0.8	0
	6.4	1.7	3.3	1.7	3.3	0.8
	64	5.8	5	5	10.8	2.5
	640	9.2	7.5	10	13.3	1.7
F_1	0	0	0	2.5	2.5	0
	6.4	3.3	10	11.7	1.7	3.3
	64	10.8	15	15	15.8	5.8
	640	12.5	14.2	17.5	19.2	7.5
F_2	0	0	1.7	0	0.8	0
	6.4	9.2	8.3	9.2	5	5.0
	64	15	15.8	17.5	14.2	7.5
	640	19.2	17.5	19.2	22.5	5.8
F_3	0	0	0	0.8	1.7	0.1
	6.4	5.0	5.8	13.3	8.3	3.3
	64	10.8	14.2	22.5	15.8	9.2
	640	10*	10*	36*	32*	8.0*
F_4	0	0	0.8	0.8	0.8	0
	6.4	6.7	8.3	10	6.7	10
	64	15.8	20	20.8	23.3	15.8
	640	20	25	27.5	30.8	17.5
F_5	0	0	0	1.7	0	0.2
	6.4	10	6.7	13.3	11.7	6.7
	64	17.5	23	25.8	21.7	19.2
	640	51.3*	37.5*	37.5*	56.3*	25*

*Excludes 3 mice killed in moribund condition. Preparations made from the testes of these mice showed absence of primary and secondary spermatocytes, spermatids, and spermatozoa.

Source: Arthur D. Little, Inc., adapted from Bulsiewicz 1977.

TABLE 33. INCIDENCE OF CHROMOSOMAL ABERRATIONS IN SPERMATOCYTES OF PHENOL-TREATED MICE

Generation	Dosage Level (ug/kg/day)	% Chromatid Breaks	% Aneuploidy	% Polyploidy	% Associations
P	0	0.3	0.1	0.1	0.1
	6.4	0.3	0.9	1.5	1.7
	64	1.4	2.0	4.4	1.9
	640	2.5	2.5	13.4	2.2
F_1	0	0.3	0	0	0
	6.4	0.4	1.7	2.4	2.4
	64	0.9	2.5	5.8	2.4
	640	4.9	1.9	12.1	3.0
F_2	0	0	0.3	0.2	0.1
	6.4	0	2.4	3.1	1.9
	64	1.6	8.6	7.2	1.8
	640	3.6	3.8	13.1	2.3
F_3	0	0	0	0.6	0.1
	6.4	0.6	3.0	2.4	2.4
	64	0.9	13.4	6.5	2.1
	640	8.7	7.1	14.2	1.2
F_4	0	0	0.1	0	0
	6.4	0.8	3.2	4.2	2.7
	64	1.2	12.6	22.9	3.6
	640	6.6	4.7	13.9	2.6
F_5	0	0.2	0.5	0.3	0.2
	6.4	2.1	2.7	6.8	4.2
	64	1.3	21	16.5	4.7
	640	14.2	14.8	26.4	3.4

Source: Arthur D. Little, Inc., adapted from Bulsiewicz 1977.

The complications associated with the interpretation of results in successive generations do not, however, mitigate the marked increase in chromosomal aberrations seen in the parental and F_1 generations. Even if the effects at the top dose are attributed to cytotoxic effects, the increased incidences of aberrations in the two lower treatment groups are noteworthy. Chromosomal breaks in spermatogonia were 1.7% and 5.8% in the 6.4-and 64-ug/kg parental groups and increased to 3.3% and 10.8%, respectively, in the F_1 generation. There were no chromosomal breaks in controls. The incidences of chromatid breaks in spermatogonia of both the parental and F_1 generations were also increased threefold or more above background. By the spermatocyte stage, the incidence of this aberration was comparable to controls at the low dose but remained elevated two to fourfold above background in the 64-ug/kg treatment group. Similar trends were noted with other chromosomal aberrations (see Tables 32 and 33).

These findings are cause for concern; even more so, is the apparent increment in the incidence of aberrations in successive generations. Further work is needed to clarify the significance of these findings and their potential implication for humans.

c. Adverse Reproductive Effects

Although not specifically designed as a reproductive study, a study by Heller and Pursell noted no signficant effects on the reproductive capabilities of rats administered 5,000 mg phenol/l in the drinking water for three generations or 100 mg/l for five generations (Heller and Pursell 1938). However, no pathological or biochemical studies were done and results were based solely on general appearance and body weights.

Korshunov reported increased incidences of pre-implantation and early postnatal deaths among offspring of rats exposed by inhalation to 5 or 0.5 mg/m^3 phenol throughout pregnancy (Korshunov 1974).

In another study by Minor and Becker no increase in fetal resorptions or teratogenic effects in offspring of Sprague-Dawley rats injected intraperitoneally with 20, 63, or 200 mg/kg phenol on either days 9-11 or days 12-14 of gestation was observed (Minor and Becker 1971). Fetal body weight was reduced at the top dose but only in fetuses from dams treated on days 12-14 of gestation (4.64 g vs. 5.25 g for controls).

Changes in the sexual cycle of female albino rats (i.e., shortening of the estrus stage and prolongation of the diestrus stage as well as disturbances in the functional state of the ovaries) were seen in rats exposed to 5 or 0.5 mg/m^3 phenol for four hours per day for four months (Kolesnikova 1972). These effects, however, may be related to the general toxic effects of phenol exposure rather than a selective response of the reproductive system.

d. Other Toxicological Effects

Regardless of the route of administration, the LD_{50} values for the mouse, rat, rabbit, and monkey are within a narrow range (0.18-0.6 g/kg) (RTECS 1978). The cat is somewhat more sensitive to phenol (oral LDLo 80 mg/kg) due to significant metabolic differences in the manner phenol is detoxified by this species.

Disturbance of the central nervous system is the predominant toxic response to phenol regardless of mode of administration. In rats, an acute lethal dose of phenol produces initial increases in pulse and respiration which later become slow, irregular, and weak. Blood pressure, after an initial rise, falls significantly. The pupils constrict in the early stages but later dilate. Salivation may be evident and dyspnea is marked. Rats also usually exhibit twitching of isolated bundles of muscles and uncoordinated movements of the legs until shortly before death (which can occur within minutes of exposure) usually due to respiratory arrest (Deichmann and Witherup 1944).

Non-lethal exposure to phenol may result in localized tissue damage. Ingestion of concentrated phenol solutions causes severe burning of mucous membranes lining the mouth and esophagus; necrosis and hemorrhage may follow. Depending on concentration, vehicle, and duration of exposure, application to the skin may result in inflammation, discoloration, eczema, sloughing, papillomas, necrosis, or gangrene (Deichmann and Witherup 1944).

Damage to tissues of the lungs, liver, kidneys, heart, and urogenital system have been reported following prolonged exposure via oral, subcutaneous, and inhalation routes of administration. In a severe intoxication the lungs may show hyperemia, infarcts, bronchopneumonia, purulent bronchitis, and hyperplasia of the peribronchial tissues. There can be myocardial degeneration and necrosis. The hepatic cells may be enlarged, pale, and coarsely granular with swollen, fragmented, and pyknotic nuclei. Prolonged administration of phenol may cause parenchymatous nephritis, hyperemia of the glomerular and cortical region, cloudy swelling, edema of the convoluted tubules, and degenerative changes of the glomeruli (Deichmann and Keplinger 1963).

Deichmann and Oesper reported that rats tolerated up to 50 mg phenol/rat/day (based on actual water consumption data) in their drinking water for up to one year without reduced weight gain (Deichmann and Oesper 1940). Slightly higher doses, $\sim$ 55-56 mg/rat/day, did reduce weight gain and water consumption was significantly reduced from normal. No histopathological studies were done. Similarly, Heller and Pursell reported normal growth rate, food consumption, and reproduction in three generations of rats given phenol in their drinking water at a concentration of 5 g/l (Heller and Pursell 1938). At 8 g/l, many young died, but the animals did reproduce. The daily dosages of phenol were not reported nor were water consumption data. No pathological or biochemical studies were done and the reported findings were based solely on general appearance and body weights.

In an unpublished study by Dow Chemical Company, rats given 20 daily doses of 0.1 g/kg phenol by gavage were reported to exhibit "slight liver and kidney effects," while rats which received 20 daily doses of 0.05 or 0.01 g/kg phenol reportedly demonstrated none of these effects (Dow Chemical Company 1976). In a subsequent series of tests, rats received 135 doses of either 0.1 or 0.05 g/kg phenol by gavage over a six month period. The growth of the rats was reported to be comparable to that of the controls. "Very slight liver changes and slight to moderate kidney damage" were reported in rats given 0.1 g/kg phenol, while administration of 0.05 g/kg of phenol was reported to result in only "slight" kidney damage.

The earlier studies by Deichmann and Oesper (1940) and Heller and Pursell (1938) administered phenol in small doses over the course of a day in drinking water, allowing metabolism and subsequent detoxification to occur. Thus, dosing regimen as well as lack of histopathological evaluation in these earlier studies may account for the lack of kidney and liver pathology in these earlier reports in contrast to the results of the Dow study.

In another Deichmann study rabbits, guinea pigs, and rats were exposed to phenol vapor at concentrations ranging from 0.1 to 0.2 mg/l (= 100-200 mg/m^3) for seven hours daily, five days per week (Deichmann et al. 1944) . Rabbits exhibited no toxic reactions after 63 exposures over a period of 88 days but examination of the lung showed widespread confluent lobular pneumonia and organization resembling granulation tissue. Peribronchial tissue was often hyperplastic and inflamed. Endothelial hyperplasia occurred in the pulmonary vessels. Upon examination of the lungs guinea pigs showed similar results but overt signs of toxicity were apparent in this species after only three to five exposures. In contrast rats were reported to show no signs of toxicity or lung damage after 53 days of exposure.

4. Human Studies

As is the case with most experimental animals, an acute lethal dose of phenol in man causes central nervous system disturbances together with peripheral dilation of blood vessels, an apparent direct effect on cardiac contractility and excitability, a failure of the vasomotor center leading to hypertension, cardiac irregularities, general tremors, convulsions, and unconsciousness or coma; death results from respiratory arrrest (Haddad et al. 1979, Stajduhar-Caric 1968, and Deichmann and Keplinger 1963). The average lethal dose in man is estimated to be between 5 and 40 grams for a 70-kilogram man, i.e., 70 to 570 mg/kg. Deichmann and Keplinger, however, report that a dose as low as one gram of phenol may be lethal (Deichmann and Keplinger 1963).

Severe chronic poisoning with phenol causes digestive disturbances, such as excess salivation, vomiting, diarrhea, anorexia, and central nervous system effects including headache, fainting, and vertigo (Deichmann and Keplinger 1963).

Chronic human exposure to low levels of phenol was examined by Baker (Baker et al. 1978) after a large phenol spill contaminated well water in a town in southern Wisconsin. Most familes continued to drink well water until an unusual taste or odor was noted. Based on water testing data for the two months following the spill and data on water preference histories obtained from impacted individuals interviewed seven months after the spill, the approximate daily oral dose was estimated to be between 10 and 240 mg. However, since phenol at concentrations of 1 mg/l imparts an unpleasant taste to water, the above range may overestimate actual ingestion. This dose range does not take into consideration skin absorption from bathing in contaminated water. Urinalyses six months after the spill revealed no significant differences in excretion of phenol between the exposed population and a control group (12 ± 12 SD and 12 ± 11 mg/l, respectively). Results of a questionnaire administered seven months after the spill revealed significant differences in the incidence of diarrhea, mouth sores, and burning of the mouth in the exposed population compared to controls.

Recently, Truppman and Ellenby reported the occurence of cardiac arrhythmias during chemical face peeling with phenol formulations (Truppman and Ellenby 1979). In 43 consecutive chemical face peels, electrocardiograms were monitored. Ten patients developed arrhythmias. The incidence was apparently not related to use of other drugs before or during the procedure, the use of nasal oxygen flush, the type of phenol formulation, age, or previous history of arrhythmias. The incidence of arrhythmias, however, did appear to be associated with the size of the area peeled and the duration of the procedure. The authors suggest that in patients who had the procedure completed more rapidly, higher blood levels of phenol were achieved; this postulate was not confirmed by chemical analyses of blood, however. There was no indication of how large a dose of phenol the patients actually absorbed, but it can be presumed that considerable absorption did occur.

Neonatal jaundice has also been recently linked to the use of phenolic disinfectant detergents in hospital nurseries (Wysowski et al. 1978, Doan et al. 1979). Wysowski evaluated two separate epidemics of neonatal hyperbilirubinemia. The first epidemic in a New Jersey hospital coincided with the use of a phenolic detergent at greater than recommended concentrations for cleaning the nursery and equipment including basinets and mattresses. To counteract an increased incidence of diarrhea believed to be a hospital-acquired infection, the phenolic detergent was mixed at two to four times recommended strength for a vigorous cleaning of the nursery. Over the next two days a cluster of six cases of severe idiopathic hyperbilirubinemia requiring exchange transfusions occurred. In the second case, an increased incidence of neonatal hyperbilirubinemia seemed to coincide with normal use of phenolic detergents in a Wyoming hospital during a period when the ventilation system was out of order. When the ventilation system was corrected and use of the phenolic disinfectant detergent was discontinued, the percentage of neonates with bilirubin levels ≥ 12 mg/100 ml fell from an average of 28% to 8-9%, or approximately the percentage experienced prior to the use of the phenolic detergent.

In another study, blood microbilirubin levels from 3-day-old infants from two nurseries were analyzed. Analysis of blood indicated a small but significantly greater microbilirubin level in neonates maintained in the nursery in which a phenolic detergent was used (Doan et al. 1979).

B. EXPOSURE OF HUMANS

1. Introduction

Although phenol is widely distributed in the environment, it does not persist long or at all in the free form. Thus, in addition to the normal endogenous production of phenol in humans, exposure to phenol occurs in certain limited situations. The biological half-life of phenol in humans is short (3-5 hours) following uptake by inhalation or dermal absorption (see Section V-A). This section will describe known exposure routes and, when possible, will quantify such exposures.

2. Ingestion

a. Drinking Water

The drinking water criteria for phenol is proposed to be set below the threshold level for organoleptic properties of chlorophenols formed during chlorination (1.0 ug/l) (U.S. EPA 1980). In a survey of 110 raw water supplies, the National Organic Monitoring Survey reported only two positive samples of phenol (no concentrations given) and no presence in finished drinking water (U.S. EPA 1978). Raw water in the lower Mississippi River averaged around 1.5 ug/l with a maximum of 7.0 ug/l of phenol (NCWQ 1975). Surface water concentrations were measured as high as 10 mg/l in 1971 but are usually less than 0.1 mg/l (U.S. EPA 1980), as described in Pathway #1, Monitoring Data (Section IV). Groundwater levels in southern Wisconsin in the vicinity and beyond the reaches of a large phenol spill were 0.21-1,130 mg/l and 0.001-0.1 mg/l, respectively.

Table 34 presents the reported concentrations of phenol in various aquatic systems and the resulting human exposure levels, assuming ingestion of 2 l/day. The highest exposure levels were associated with the phenol spill and ingestion of untreated surface water (at 10 mg/l). The background-level phenol concentrations resulted in exposure levels similar to those associated with the more typical surface water upper-limit concentrations. Because virtually no other data were available on groundwater levels of phenol, it is not possible to know how representative the Wisconsin levels are of well concentrations in general.

TABLE 34. EXPOSURE LEVELS RESULTING FROM INGESTION OF PHENOL IN FOOD AND WATER

Source	Phenol Concentration (mg/l)	Daily Exposure Level (mg)
Water		
• finished drinking water	ND[1]	negligible
• raw drinking water	0.007	0.014[2]
• unfinished surface water	10(max); 0.1 (more typical)	20; 0.2[2]
• well water 1) maximum	1,130	2,260[2]
2) initial	0.21 - 3.2	0.42 - 6.4[2]
3) calculated exposure level from case study (Baker *et al*. 1978)	-	10 - 240[2]
Food	(mg/kg)	(mg)
• smoked summer sausage	7	0.1[3]
• smoked pork belly	28.6	0.6[3]
• fish	50(max); 16(mean)	1.1; 0.34[4]

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[1] Non-detectable

[2] Assuming ingestion of 2 l/day

[3] Assuming ingestion of 20 g/day (USDA 1978)

[4] Assuming ingestion of 21 g/day (USDA 1978)

Table 34 also presents the more representative exposure levels (taking into account the fluctuation in phenol levels over time) calculated for the highest exposure group in a post-spill study of the incident (Baker et al. 1978). The results of this study are discussed in more detail in Sections V-A and VII-A.

b. Food

Phenol has occasionally been found in food items, although a comprehensive diet analysis has not been conducted. Lustre and Issenberg found 7 mg/kg phenol in smoked summer sausage and 28.6 mg/kg in smoked pork belly, the phenol presumably originating from the wood used in processing the meat (Lustre and Issenberg 1970). In addition, while only limited data are available, mean concentrations of 16 mg/kg phenol in fish have been reported, while maximum values were 50 mg/kg (see Section V-B). Table 34 also presents food concentrations and exposure levels.

The maximum exposure level resulting from ingesting all three food items (assuming the maximum smoked meat and fish levels) is approximately 2 mg/day. Assuming the lower flesh concentrations would result in a combined exposure level of 2.5 mg/day, a maximum consumption of fish (about 200 g/day--NMFS 1978) would result in a maximum exposure to phenol at 10 mg/day (for maximum concentration). Likewise, maximum consumption of smoked meat results in an exposure level of 6 mg/day (for maximum concentration). Exposures of these magnitudes derived from ingestion of these particular products are expected to be limited to small subpopulations; however, monitoring data for phenol in a broad spectrum of food products are not available. Until monitoring is conducted it is not possible to know the size of the population exposed to comparable or higher phenol levels in food.

c. Products Containing Phenol

Phenol is used in several consumer products including Cepastat ® mouthwash and lozenges (1.45% phenol), Chloraseptic® mouthwash (1.4% phenol), and Chloraseptic® lozenges (32.5 mg/total phenol/lozenge) (U.S. EPA 1979). Use of the mouthwash (60 ml/day) would result in the intake of 870 mg/day, if ingested; however, only a small amount of this volume would be swallowed and/or absorbed. Assuming a 10% retention of the mouthwash at a maximum, the actual intake would be 87 mg/day. Use of the lozenges at the recommended dosage of eight per day would result in an exposure of 260 mg/day for the duration of use of the product. Table 35 presents the estimated exposure levels for phenol-containing products.

TABLE 35. EXPOSURE LEVELS RESULTING FROM USE OF MEDICINAL PRODUCTS

Source	Phenol Concentration[1]	Daily Exposure Level (mg)
Ingestion:		
Cepastat® mouthwash	1.45%	87[2]
Chloraseptic® mouthwash	1.4%	
Cepastat® lozenges	32.5 mg/lozenge	260[3]
Dermal Absorption:		
Noxzema® face cream	0.5%	12.5[4]

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[1] All concentrations from U.S. EPA 1979.

[2] Assuming 60 ml used per day, 10% retention

[3] Assuming ingestion of eight lozenges per day

[4] Assuming use of 5 gm/daily and 50% absorption

The number of persons actually using these commonly available products is unknown. The population associated with these exposures, therefore, is expected to be large; however, the products are for use during illness and thus would probably not be used daily for extensive periods.

Fishbeck examined the urine of a subject ingesting Chloraseptic® lozenges every two hours for a total of eight doses (Fishbeck et al. 1975). The free phenol level in the urine peaked at 10.0 mg/l in the third eight-hour period after starting the doses. The total phenol content of the urine returned to baseline within 48 hours after ingestion of the last lozenge. These results confirm the rapid clearance of phenol in man.

3. Inhalation

The most significant exposure through inhalation of phenol is primarily confined to occupational settings. Data found for ambient air levels of phenol were discussed in Section III. Deimel and Gableski reported 0.02-0.3 mg/m^3 phenol in air along highways in Germany (Deimel and Gableski 1973). Assuming an inhalation of 20 m^3 of air/day, a maximum exposure of 6 mg phenol/day would result. It is unknown how valid these exposure levels are for U.S. cities; however, in industrialized areas with high traffic volume, comparable U.S. levels may exist.

The use of phenol in the laboratory for purposes such as organic synthesis and plastics formulation would also result in exposure to humans. Assuming that work was being conducted in a small, poorly ventilated room in which air saturation by phenol would be reached--a worst case--an air concentration was estimated. Based on Henry's law, phenol in a beaker would generate 9.4 mg of phenol/m^3 of air. Assuming an inhalation rate of 7 m^3 air in 8 hours, the resulting exposure level would be 75.2 mg of phenol (Arthur D. Little, Inc., estimate).

4. Dermal Absorption

Phenol is found in various cosmetics and skin care products (U.S. EPA 1979). Most of these products are used for temporary relief of skin problems, such as poison ivy or burns; however Noxema Medicated® (Noxell) and similar products may be used on a daily basis. For purposes of estimation, the use of 5 grams of such a product per day is assumed and would result in the application to the skin of 0.25 mg phenol per day. Assuming 50% of this would be washed off, and considering the rapid dermal absorption of phenol described in Section V-B, then 12.5 mg of phenol is a rough estimation of the amount absorbed per day (Arthur D. Little, Inc., estimate).

The use of face peels containing phenol has been associated with an increased incidence of arrhythmias. Although this use apparently represents a source of human exposure, the dose received cannot be readily calculated.

5. Exposure Scenario Estimates

For the purpose of comparing exposure levels from different types of exposure and total levels resulting from combined exposures, five exposure scenarios were fabricated (see Table 36). A worst case scenario (Scenario #1) combining the greatest exposure levels resulted in a daily exposure of approximately 520 mg of phenol. Three special-case exposure scenarios for subpopulations exposed to contaminated groundwater (Scenario #2), treated with phenol-containing medicinal products (Scenario #3), and working in a laboratory (Scenario #4) resulted in exposure levels of approximately 250, 350, and 80 mg/day, respectively. The inclusion of daily use of face cream increased these levels to 260, 370, and 95 mg/day respectively. Finally a general population scenario (Scenario #5) had an associated exposure level of 7 mg/day.

C. OVERVIEW AND CONCLUSIONS

This section has discussed the effects on humans of exposure to phenol and their level of exposure.

1. Effects

Phenol is readily absorbed from all routes of entry, distributed throughout the body, and metabolized and excreted from the body in rather rapid order. The half-life of phenol in man is approximately 3.5 hours. Phenol is also produced endogenously by the degradative action of bacteria in the gut on tyrosine.

Acute lethal values for phenol are all within an order of magnitude, regardless of the route or species and, for the most part, are in the 200-700 mg/kg range. The cat appears to be the most sensitive species (oral LDLo 80 mg/kg), probably as a result of significant metabolic differences in the manner phenol is detoxified in this species. Slight to moderate kidney damage and slight liver changes have been reported in rats given 135 daily doses of 100 mg/kg phenol by gavage. Similar treatment with 50 mg/kg produced slight kidney damage after 135 doses but not after 20 doses. Rats, however, have been able to tolerate much larger doses in drinking water (56 mg/rat/day or $\sim$ 280 mg/kg for a 200-g rat), probably due to its rapid metabolism as well as the intermittent nature of dosing in contrast to exposure by gavage.

There are no indications that phenol is carcinogenic by the oral route, but it does appear to possess tumor-promoting activity. Skin application of phenol is tumorigenic in sensitive strains of mice but not in standard inbred strains of mice. The tumorigenic activity of phenol appears to be associated with its irritancy and subsequent skin hyperplasia.

Phenol has been shown to induce lethal mutations in fruit fly (Drosophila) and to significantly increase the incidence of chromosomal effects in a dose-related manner in spermatogonia and spermatocytes of mice given phenol by gavage at dosage levels as low as 6.5 ug/kg/day. Furthermore, the data suggest incremental increases in the incidence of

TABLE 36. EXPOSURE SCENARIOS INVOLVING CONTACT WITH PHENOL

Scenario	Exposures	Exposure Level (mg /day)
Scenario #1		
Worst Case (very small subpopulation affected)	Ingestion of contaminated well water	240[1]
	Ingestion of contaminated fish	1.1
	Ingestion of throat lozenges	260[1]
	Use of face cream	12.5
	Inhalation along highway[2]	6[3]
	Total	519.6
Scenario #2		
Subpopulation in area with contaminated groundwater (small subpopulation affected)	Ingestion of contaminated well water	240[1]
	Ingestion of contaminated fish	1.1
	Inhalation along highway	6[3]
	Total	247.1
Scenario #3		
Subpopulation being treated with medicinal products containing phenol (small subpopulation affected)	Ingestion of throat lozenges	260[1]
	Ingestion of mouthwash	87[1]
	Ingestion of raw drinking water	0.014
	Ingestion of contaminated fish	1.1
	Inhalation along highway	6[3]
	Total	354.114
Scenario #4		
Subpopulation exposed in laboratory (small subpopulation affected)	Inhalation of laboratory air[4]	75.2
	Ingestion of raw drinking water	0.014
	Ingestion of contaminated fish	1.1
	Inhalation along highway	6[3]
	Total	82.34
Scenario #5		
General population (majority of population affected)	Ingestion of raw drinking water	0.014
	Ingestion of contaminated fish	1.1
	Inhalation along highway	6[3]
	Total	7.114

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[1] Short-term exposure

[2] Although inhalation and ingestion are different exposure routes, the levels have been combined to provide an idea of the overall magnitude of exposure of each hypothetical subpopulation

[3] Based on estimate from German study. It is unknown how realistic this is for the U.S.

[4] Assuming daily use of phenol.

Source: Arthur D. Little, Inc., estimates.

chromosomal aberrations occur in consecutively treated generations. However, the treatment schedule utilized and the lack of data reporting prevent assessment of the significance of this finding. No indications of teratogenicity have been found.

The lowest reported oral lethal dose in man is one gram of phenol, but the majority of lethal values are in the 5- to 40-gram range. An acute lethal dose of phenol results in central nervous system disturbances together with peripheral vasodilation leading to sudden collapse and unconsciousness. Death is due to respiratory arrest. Ingestion of non-lethal amounts of phenol can result in burning in the mouth, mouth sores, headache, vomiting, diarrhea, back pain, paresthesia, and production of dark urine (probably from oxidation products of phenol). Recent reports have also linked phenol to the production of cardiac arrhythmias during chemical face peeling procedures.

Aside from the issue of its mutagenicity, the rapid clearance of phenol from the body, its relatively high lethal dose, and the fact that small amounts of phenol are produced endogenously indicate that man can handle levels normally present in U.S. drinking water with no untoward effects. Further work needs to be done to validate the single report of increased chromosomal aberrations in phenol-treated mice and, in particular, to clarify the finding of increased numbers of aberrations in consecutively treated generations of mice.

2. Exposure

There are numerous uncertainties involved in estimates of exposure to phenol, primarily due to lack of monitoring data. Based on the limited information available, the use of phenol-containing products, especially mouthwash and lozenges, represents the largest consumer exposure, although presumably on a short time scale. Ingestion of contaminated well water may result in an equivalent short-term exposure. Other water supplies, even untreated surface water, would contribute to a very small exposure level through ingestion. Laboratory workers are a subpopulation potentially exposed to levels equivalent to use of phenol-containing mouthwash; however, it is assumed this exposure would occur over a longer period of time. Ingestion in food and dermal absorption from cosmetics may contribute to a more continual exposure for this subpopulation. Ingestion of fish or smoked meat and inhalation along highways may each represent an exposure of 10 mg/day, using worst case assumptions.

Special note should be made of the potential conversion of phenol during chlorination to lower chlorinated phenols. Although phenol is eliminated during this process, more harmful compounds, including known carcinogens, may result. A separate exposure assessment considers the environmental distribution of effects of and exposure to three commonly detected chlorophenols--2-chlorophenol, 2,4-dichlorophenol, and 2,4,6-trichlorophenol (Scow et al. 1980).

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SECTION VI.

AQUATIC BIOTA EFFECTS AND EXPOSURE

A. EFFECTS ON AQUATIC BIOTA

1. Introduction

This section provides experimental information on the levels of phenol at which the normal behavior and metabolic process of aquatic organisms are disrupted. Although phenol has been studied fairly extensively for its acute toxic effects, bioassay results are not often consistent, even for a single species. Tests conducted under static conditions are normally less reliable than continuous-flow experiments because there is less control of phenol concentrations. However, continuous flow conditions may be be suitable for testing certain kinds of organisms, such as fish larvae and free-floating invertebrates.

In both kinds of bioassays, phenol concentrations are often determined determined nominally (i.e., by diluting a measured amount of phenol) instead of by direct periodic measurement during the bioassay. Nominal determination of concentrations does not account for phenol evaporation, adsorption onto particles or walls of test tank, or absorption by test organisms, and so may produce overestimates of lethal and sublethal levels. In a flow-through experiment where the phenol levels were monitored (Kristoffersson *et al*. 1973), the authors twice had to nearly double the concentration in the influent water to maintain the desired concentration in the test aquarium. They attributed the loss of phenol to oxidation by dissolved oxygen or bacterial degradation.

Water temperature has been demonstrated to affect the sensitivity of aquatic animals to phenol, largely as a result of its influence on metabolic rate. Other factors such as pH and hardness, which are known to alter the toxicity of other chemicals, have not yet been studied extensively for phenol. In addition, the species and the developmental stage of the test organisms must be considered. Since some species and stages may be more sensitive to phenol than others, it may not be appropriate to compare the data from unrelated studies.

2. Freshwater Organisms

a. Chronic and Sublethal Effects

Low levels of pollutants which remain for extended periods are generally considered to represent "normally" polluted conditions in natural waterways. Under these circumstances aquatic biota may become acclimated to the pollutant, or they may exhibit certain behavioral or

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physiological responses. Prolonged exposure, even to low concentrations of phenol, could ultimately result in mortality. Even if fish are not killed by long-term exposure to phenol, the survival of local populations may be endangered.

A summary of sublethal and chronic effects data for vertebrates and invertebrates is presented in Table 37. Daphnids were the most sensitive species tested; however, the increased molting and growth rate reported for *Daphnia pulex* cannot necessarily be considered a detrimental effect. The grass frog (*Rana temporaria*) is the only non-piscine vertebrate for which toxicity data were found. According to the data of Kaufmann (1977), the critical stages for susceptibility to phenol in frog development are the tail-bud stages and during metamorphosis.

The physiological effects in rainbow trout (*Salmo gairdneri*) described by Mitrovic and others are more pre-mortality than sublethal in nature (Mitrovic *et al*. 1968). In their experiments with freshwater worms, Alekseev and Uspenskaya observed a general sequence of effects preceding death from phenol toxicosis (Alekseev and Uspenskaya 1974). The typical progression was as follows: normal swimming or burrowing activity, increased locomotor activity, swimming worms sink to floor of test aquarium, curling of body or tail, and convulsive twitching followed by a lack of response to mechanical stimulation. The phenol concentrations producing these effects ranged from approximately 100 to 1,000 mg/1.

Numerous species of algae have been bioassayed for susceptibility to phenol, with most of the experimentation performed by Kostyaev (Kostyaev 1973). The lowest concentration at which sublethal effects have been reported is 8 mg/1, which completely inhibited photosynthesis in *Uroglenopsis divergens*. Kostyaev found that green algae generally were most resistant, while chrysophytes were the most sensitive. In some cases, low concentrations of phenol stimulated photosynthesis, as in the case of *Chlorella* exposed to 10-40 mg/1 (Lukina 1970). Higher concentrations, however, had the reverse effect, and still higher levels may inhibit respiration as well. For more details on phenol toxicity to algae, see Table 13 in Buikema *et al*. 1979.

b. Acute Effects

Acute toxicity is defined as toxicant-induced mortality over a short period of time, generally within 96 hours. Although fish in natural waterways are more likely to be exposed to lower concentrations which may result in chronic or sublethal effects, industrial discharges and spills can temporarily result in levels high enough to cause fish kills (see Section VI-B).

TABLE 37. CHRONIC AND SUBLETHAL EFFECTS ON FRESHWATER ORGANISMS

Concentration (mg/l)	Species	Test Duration	Effects	References
0.1	*Daphnia magna*	2 weeks	Decreased reproductive rate	Luferova and Flerov 1974
0.1	*Daphnia pulex*	2 weeks	Increased molting and growth rate	Luferova and Flerov 1974
0.5	Grass frog (*Rana temporaria*)	-	35% survival of embryos; survivors undeveloped, with malformed tails	Kaufmann 1977
2.6	Fathead Minnow	--	Chronic value	Holcombe et. al., 1980
3.1	*Daphnia magna*	-	Chronic value	U.S. EPA 1978
5	Northern Pike	5 hours	Loss of balance	Kristoffersson *et al*. 1973
5	*Daphnia longispina*	2 weeks	Decreased fecundity	Luferova and Flerov 1971
6.5, 6.9	Rainbow trout (*Salmo gairdneri*)	7 days	Lesions at base of fins, excessive mucous secretions on skin and gills, inflamed and bleeding gills with damaged lamellae, swollen spleen, kidney, and liver	Mitrovic *et al*. 1968
25	Bream (*Abramis brama*)	-	Hatching delayed by 1 day	Volodin *et al*. 1966
40	Russian sturgeon, stellate sturgeon	12 days	No development of pigmentation in prolarvae	Shmal'gauzen 1974
400	Clams (*Dreissena polymorpha*) (*Sphaerium corneum*)	4-5 days	Decreased filtration (feeding) rate	Smirnova 1973

The acute effects of phenol on freshwater finfish have been studied for at least thirteen species, resulting in a fairly reliable data base which has been compiled and condensed in Table 38. It should be noted that the LC_{50} values given were derived under a variety of conditions. Such factors as exposure period (between 24 and 96 hours), age of test fish, difference in certain water parameters, and bioassay type (static or flow-through) may account for some of the variation in a given species' sensitivity. (Factors contributing to variability in phenol toxicity and fish sensitivity are discussed in greater detail in Section VI-A4).

Reported LC_{50} values for phenol range from 5.0 mg/l for the juvenile rainbow trout (*Salmo gairdneri*) to 200 for the goldfish (*Carassius auratus*). The data are insufficient to attempt to identify the most sensitive families; the most frequently tested particular species are likely to have the widest ranges of reported LC_{50}'s. However, it should be noted that salmonids are normally among the most sensitive of the species bioassayed.

One study which is occasionally referred to in the literature reports a very low lethal level of 0.08 mg/l for a "minnow" species (Symons and Simpson 1938). The phenol was present, however, in a mixed waste including other unidentified toxic substances; therefore, this concentration should not be used to represent phenol's effects (EIFAC 1973).

LC_{50}values for freshwater invertebrates are summarized in Table 39. As a great many species have been tested for sensitivity to phenol, this list provides only a sample which represents the range of LC_{50}'s reported. Both the lowest (<1.5 mg/l) and highest (1,840 mg/l acute values were determined by Alekseev (Alekseev 1973) for aquatic insects, *Baetis* species and *Mideopsis orbicularis*, respectively. Common names were not found for these invertebrates. Of the eleven species of worms tested by Alekseev and Uspenskaya (Alekseev and Uspenskaya 1974), benthic worms (habituated to a lack of oxygen) were the most resistant to phenol. The species which lived in bottom detritus above the silt were somewhat more sensitive, while those which swam in the water were generally the most susceptible. For a more complete listing of acute toxicity data for freshwater invertebrates see Table 14 in Buikema *et al*. 1979.

3. Marine Organisms

The data base on the toxic effects of phenol on seawater biota is limited to acute toxicity bioassays on two fish and two mollusc species. In a 60% (salinity 20^{o}/oo) seawater solution, rainbow trout (*Salmo gairdneri*) exhibited median mortality at 5.2 mg/l phenol (Brown et al. 1967). Since resistance decreased as the proportion of seawater increased from 0% to 60% it could be assumed that the LC_{50} is lower in 100% seawater ($\sim$30 ^{o}/oo) salinity. Nunogawa and others determined a 96-hour LC_{50} of 6.0 mg/l for the mountain bass (*Kuhlia sandvicensis*), a species native to Hawaiian waters (Nunogawa *et al*. 1970).

TABLE 38. ACUTE TOXICITIES (LC$_{50}$) FOR FRESHWATER FISH

Range of LC$_{50}$ (mg/l)	Species
5.0-11.6	Rainbow trout (*Salmo gairdneri*) ; juvenile
11.5-60.0	Bluegill sunfish (*Lepomis macrochirus*)
11.7	Brook trout (*Salvelinus fontinalis*)
16.7	Channel catfish (*Ictalurus punctatus*)
19.0	Mozambique mouthbrooder (*Tilapia mossambica*)
22.0-63.0	Molly (*Mollienesia latipinna*)
24.0-67.5	Fathead minnow (*Pimephales promelas*)
26.0	Mosquitofish (*Gambusia affinis*)
31.0-39.19	Guppy (*Poecilia reticulatus*)
31.5	Walking catfish (*Clarias batrachus*)
33.3[1]-200.0	Goldfish (*Carassius auratus*)
35.0-129.0	Golden shiner (*Notemigonius chrysoleucas*)
36.3	Flagfish (*Jordanella floridae*)

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[1] Gersdorff 1939

Source: Compiled from Table 1, U.S. EPA 1980, except where noted.

TABLE 39. ACUTE TOXICITIES (LC_{50}) FOR FRESHWATER INVERTEBRATES

Range of LC_{50} (mg/l)	Species	Reference
<1.5	*Baetis* sp.	Alekseev 1973
7-100	*Daphnia magna*	
14	*Daphnia longispina*	
15-78	Isopod (*Asellus aquaticus*)	
18-93	*Daphnia pulex*	
57	Cladoceran (*Polyphenus pediculus*)	
78	Conchostracan (*Lynceus brachyurus*)	
94	Snail (*Physa heterostropha*)	
100	Worm (*Euplanaria lugubris*)	Alekseev and Uspenskaya 1974
108	Copepod (*Mesocyclops leukarti*)	
122	Copepod (*Cyclops vernalis*)	
150	Worm (*Mesostoma ehrenbergii*)	Alekseev and Uspenskaya 1974
205-300	Rotifer (*Philodina acuticornis*)	
260-320	Snail (*Physa fontinalis*)	
341-381	Annelid (*Aelosoma headleyi*)	
350	Snail (*Limnaea stagnalis*)	
351-391	Snail (*Nitrocris sp.*)	
520	Worm (*Lubriculus variegatus*)	Alekseev and Uspenskaya 1974
780	Clam (*Sphaerium corneum*)	
1,280	Worm (*Helobdella stagnalis*)	Alekseev and Uspenskaya 1974
1,840	*Mideopsis orbicularis*	Alekseev 1973

Source: Compiled from Table 2 in U.S. EPA 1980, except where noted.

The larvae of the Atlantic oyster (Crassostrea virginica) and the hardshell clam (Mercenaria mercenaria) have been bioassayed by Davis and Hidu (1969). The resulting 48-hour LC_{50} values were 58.3 and 52.6 mg/l, respectively.

4. Factors Affecting the Toxicity of Phenol

Of the many parameters that are controlled in toxicity bioassays, water temperature is the factor that has been most frequently tested for its effects on phenol toxicity. In experiments with fathead minnows (Pimephales promelas), Ruesink and Smith found that the 96-hour LC_{50} decreased from 36 to 24 mg/l as the temperatures increased from 15 to 25°C (Ruesink and Smith 1975). Brown and others observed the opposite effect in rainbow trout (Salmo gairdneri); the 48-hour LC_{50} at 18°C was almost twice that at 6°C (Brown et al. 1976). However, the response period of the fish decreased with the rise in temperature so that death occurred more quickly at the higher temperatures. Moreover, the expected trend might have reappeared if higher temperatures (>18°C) had been tested. In a bioassay which allowed brook trout (Salvelinus fontinalis) a selection of temperatures from 4 to 29°C, fish exposed to phenol consistently chose areas of lower temperature than controls. The difference in temperature selection was particularly significant in groups exposed to 7.5 and 10.0 mg/l phenol (Miller and Ogilvie 1975).

In their bioassays with freshwater worms, Alekseev and Uspenskaya found that the overall resistance of the worms increased as the water temperature declined (Alekseev and Uspenskaya 1974). However, the maximum tolerated concentrations (at which no mortalities occurred) were lowest at the most extreme temperatures, 2 and 28°C. The results from all the studies suggest that, to a certain degree, the effects of variations in temperature are species- or at least group-specific.

In order to determine the effects of salinity variations on the resistance of a salmonid species, Brown and others exposed rainbow trout to phenol in water solutions containing from 0 to 60% seawater (0 °/oo to 20 °/oo salinity) (Brown et al. 1967). At 15°C the 48-hour LC_{50} steadily decreased from 9.3 mg/l in fresh water to 5.2 mg/l in 60% (~20 °/oo) seawater. Presumably the LC_{50} would have been yet lower in 100% (~33 °/oo) seawater. The authors pointed out that the greatest hazard to migrating salmonids with respect to phenol effects would be at the seaward end of their passage due to increased toxicity at higher salinity. Further study in this area should consider the ability of salmonid species to acclimate to saline water in the presence of phenol.

The only information on pH effects on phenol toxicity was from a study by Flerov and Luk'yanenko (1966). Crucain carp were more sensitive to phenol at pH extremes, but within an intermediate range, toxicity levels did not vary. It is likely that pH extremes are harmful to fish independent of any effects they might have on phenol toxicity.

Acclimation may be an important factor in the ability of fish and invertebrate populations to survive chronic exposure to phenol. Flerov found that guppies which had been raised in low concentrations of phenol for three generations were five times as resistant to phenol as unacclimated stock (Flerov 1971).

B. EXPOSURE OF AQUATIC BIOTA

Fish-kill data indicate that phenol containing wastes sometimes reach concentrations in aquatic systems high enough to have lethal effects on aquatic organisms. Unfortunately, monitoring data on ambient phenol levels in surface waters are too limited to support non-speculative conclusions on the aquatic exposure of biota to phenol. No information on exposure levels to wildlife in terrestrial systems was available.

Monitoring data for phenol levels in surface waters are quite limited (see Section IV-Pathway #1). The data provided by STORET on phenol levels in ambient waters of U.S. river basins during 1978-1980 amount to less than 600 observations, most of them remarked data, indicating that phenol is an infrequently monitored chemical. The only major basins with more than 50 observations were the Southeast, Ohio River, Lower Mississippi, and Pacific Northwest regions. The river basins with the highest unremarked concentrations were the Tennessee and Ohio River basins. The maximum values were, respectively, 6,794 ug/l and 5,900 ug/l. In both basins these maximums were most likely unusual observations and could not be assumed to be representative of the entire basin due to insufficient data or a high standard deviation in the available data.

Table 40 summarizes the frequency distribution of phenol concentrations over ranges from <0.99 to >1,000 ug/l. In addition to observations reported in 1978 through 1980, earlier reported ranges are also included separately. The data were not combined because of the unreliability of some of the earlier measurements. Unremarked data tended to be reported at less than 10 ug/l (72%) and remarked data between 1 ug/l and 100 ug/l (89%).

Ambient surface water concentrations from sources other than STORET were also few and inconclusive. The highest U.S. phenol concentration reported (also in STORET) of 142 ug/l in the Delaware Estuary is actually for phenolics (Faust _et al_. 1975). Therefore, if other concentrations in the STORET data base also cover other phenols, one would suspect that phenol itself is present at even lower levels than reported. It is not possible, however, to check the analytical techniques for each observation, so it is conservatively assumed that all STORET and other levels measure only phenol.

Judging by the results of the EXAMS concentration simulations for a lake and river system (see Section IV-Pathway #1), it appears unlikely that even raw waste levels are high enough to lead to, under conditions of continuous discharge, phenol levels of a magnitude greater than that

TABLE 40. FREQUENCY DISTRIBUTION OF PHENOL (TOTAL) CONCENTRATIONS IN AMBIENT SURFACE WATER[1]

1970-1977

	Remarked Data					Unremarked Data				
Year	≤0.99	1-9.99	10-99.99	100-999.99	≥1000	<0.99	1-9.99	10-99.99	100-999.99	>1000
1970	-	2	1	-	-	-	-	-	-	3
1971	-	-	-	-	-	-	-	-	-	1
1972	4	-	1	-	-	7	-	-	-	-
1973	-	-	2	8	-	-	6		-	-
1974	-	46	1	-	-	-	71	-	-	-
1975	-	87	2	-	-	-	108	6	-	-
1976	-	23	-	-	-	3	60	1	-	-
1977	3	-	10	-	-	-	10	14	-	-
Gross	7	158	17	8	-	10	255	21	-	4

1978-1980

	Remarked Data					Unremarked Data				
Year	<0.99	1-9.99	10-99.99	100-999.99	≥1000	≤0.99	1-9.99	10-99.99	100-999.99	≥1000
1978	16	66	43	-	-	7	1	-	-	-
1979	7	20	120	-	-	8	-	1	-	1
1980	38	25	240	3	2	2	11	6	3	1
Gross	61	111	403	3	2	17	14	7	3	2

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[1] Numbers at top of column are concentration ranges in units of ug/l. Numbers in body of table are numbers of observations reported within each range.

Source: U.S. EPA (1980d).

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for typical surface water. However, it was not possible to identify and/or estimate the quantity of phenol generated by each waste stream and it is possible that other industries may contribute higher levels.

C. CONCLUSIONS

1. Effects

The lowest concentration of phenol at which toxic effects have been reported is 0.1 mg/l, in *Daphnia magna*. The lowest acute level was for a species of *Baetis* (insect), a 48-hour LC_{50} of <1.5 mg/l. Rainbow trout was the most sensitive fish tested, with LC_{50} values as low as 4.2 mg/l. The grass frog was the only non-piscine vertebrate tested; lethal toxicosis in embryos was reported at 0.5 mg/l phenol.

Toxicosis was manifested in a number of effects in addition to mortality. Decreased reproductive rate and fecundity were observed in *Daphnia*, loss of balance in pike, lack of pigmentation in developing sturgeon prolarvae, delayed hatching in bream, and reduced feeding in clams. Not all effects were detrimental, however; *Daphnia pulex* grew more quickly in 0.1 mg phenol/l, and some species had higher hatching rates in very low concentrations of phenol.

Data on toxicity to marine organisms is extremely limited, although the toxic levels reported are in the same range as for freshwater species. No eco-community or population studies in either laboratory or field conditions were available.

Environmental factors may have an influence on the toxicity of phenol to aquatic life. Water temperature is the most extensively studied variable, yet studies present variations between species as to its general effects. In most cases, the organism becomes more sensitive as temperature increases, although high and low extremes appear to be the most detrimental. The anadromous rainbow trout perished at lower phenol concentrations as salinity increased, suggesting that salmonid populations are most at risk as their migrations bring them into estuaries. Crucian carp were more sensitive to phenol at pH extremes, while intermediate acidity levels did not affect toxicity concentrations.

2. Exposure

Based on available data, phenol concentrations of any significance in regard to aquatic life (see Section VI-B) are few and short-term. Under conditions of continual discharge, most reported effluent levels appear unlikely to contribute high phenol concentrations in most aquatic systems. In addition since these loadings are based on raw wastewater phenol levels, wastewater treatment would reduce them significantly before release. Examples of situations in which environmentally adverse phenol concentrations occurred and the effects of these levels on aquatic populations are discussed in Section VII.

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SECTION VII.

RISK CONSIDERATIONS

A. INTRODUCTION

The purpose of this section is to evaluate the risks of exposure of humans and aquatic biota to phenol primarily in view of the levels at which effects have been reported under laboratory conditions. Data are limited for phenol in the areas of both effects and exposure; however, several field studies were available on the impact of phenol on human and aquatic populations exposed to environmental concentrations. The following section addresses, first, risk considerations for man, and then, for other biota.

B. HUMANS

1. Statement of Risk

Humans are rarely exposed to concentrations of phenol in environmental media at levels high enough to cause adverse effects (as distinguished from laboratory studies on animals). However, one effects study reported chromosomal damage to mice at concentrations far below all other effects levels and at environmental exposure levels that a large fraction of the human population may encounter. If correct, this study suggests that further investigation of low-level effects of phenol on laboratory animals is warranted. Other effects in laboratory animals are reported at concentrations of 50 mg/kg/day and higher. Exposure levels for humans, even under special phenol-intensive conditions, are generally less than 4 mg/kg/day with few exceptions; users of phenol-containing medicinal products and people maximally exposed to almost all sources of phenol simultaneously may be exposed to levels of up to 9 mg/kg/day. Typical levels of phenol in food, drinking water, and air are far below those related to adverse effects in laboratory animals (excluding the chromosomal study). Caution should be taken, however, in drawing any final conclusions from comparison of phenol exposure levels for humans and effects levels for laboratory animals due to species and dosage differences. As is true for most substances, the significance of toxicity data obtained under experimental conditions relative to actual conditions of exposure is not well understood.

2. Discussion

Phenol is readily absorbed from dermal, oral, and inhalation routes, metabolized, and then rapidly excreted from the body. The half-life of

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phenol in man is approximately 3.5 hours. Phenol is also endogenously produced in humans and, in conjugated form, is a normal constituent of urine.

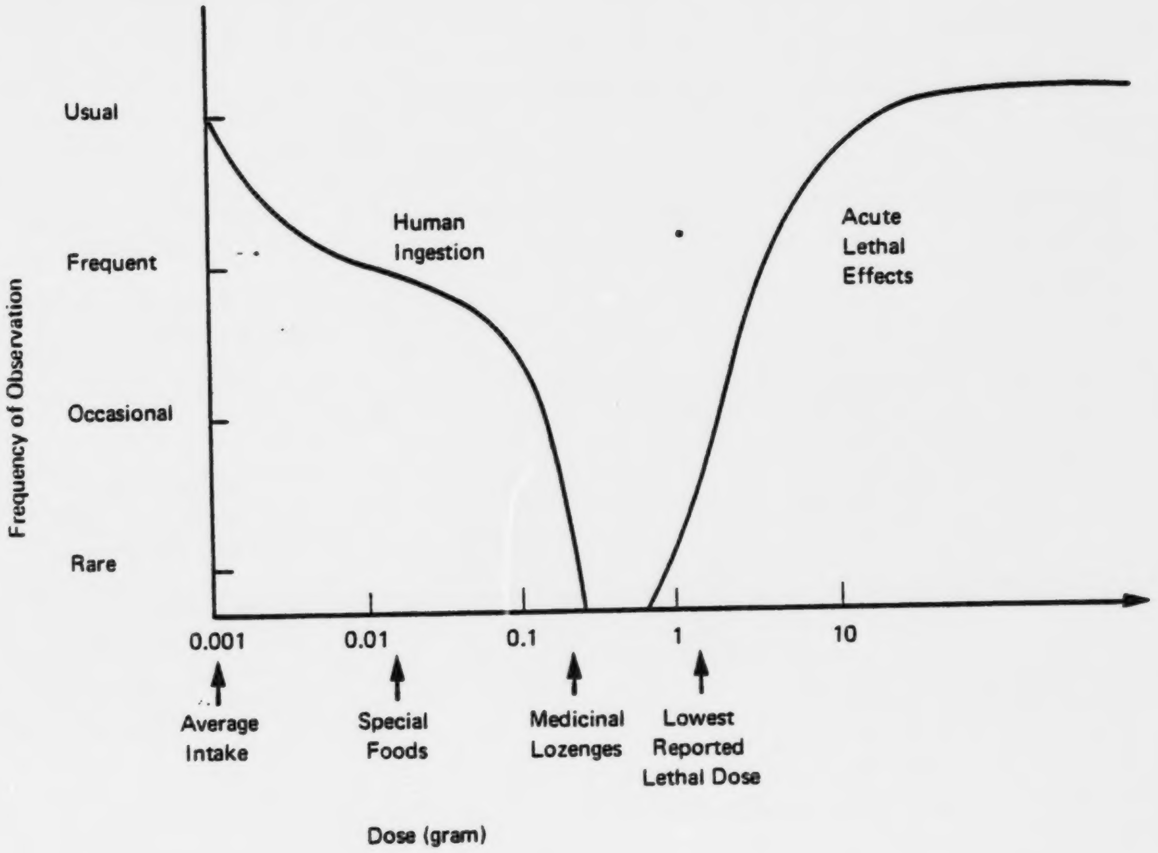
NCI recently reported that phenol was not carcinogenic in either rats or mice when administered in drinking water at concentrations of up to 0.5% by volume (NCI 1980). However, further testing has been recommended (see Section V). Phenol has exhibited some tumor-promoting activity in skin-painting studies, but the nature of these studies makes them inappropriate for the assessment of human risk by ingestion. These and other data on effects are summarized in Table 41.

Renal damage has been reported in rats at a 50-mg/kg dose of phenol daily for 4 months. This effect is unlikely to occur in man in that ingestion exposures are generally distributed throughout the course of the day and thus allow metabolic processes to rapidly clear phenol from the body.

Chromosomal aberrations have been observed in mice at very low doses (6.4 ug/kg/day). These results represent the work of one investigator (see discussion in Section V) and have not been confirmed. If these results are valid, and at present there is no reason to discount them on scientific grounds, then, they are cause for concern. Even given the uncertainties involved in the exposure estimates, and the fact that the entire dose was administered at once as opposed to over a 24-hour period, the phenol content of certain foods and phenol-containing products is sufficiently high to constitute human risk. Additionally, the study is relevant to humans for several reasons: similarities in mammalian metabolic processes in regard to phenol, the fact that the treatment method (exposure of pregnant mothers and their offspring) represents a reasonable exposure route, and the general preference of _in vivo_ studies to _in vitro_ studies. The quantification of such a risk of human genetic impairment from animal data is, at present, not feasible, and will remain so until a correlation can be demonstrated between test animals of a rather uniform genetic background and humans with wide individual variations. In addition, substantiation of the results of this study is warranted.

Table 42 summarizes exposures described more fully in Section V. It should be pointed out that these estimates are based on numerous assumptions and that there is considerable uncertainty involved. In addition, the size of the subpopulation involved is difficult to quantify. Nevertheless, it is evident that the use of phenol-containing products represents the largest single source of exposure other than occupational exposure.

Figure 17 roughly compares the doses of phenol reported for effects resulting from various exposures to the frequency of observation. The diagram expresses qualitatively the likelihood of equivalent exposure and effects levels. It is apparent that single exposures are not high enough to cause the lowest observed effect. Although the margin



Source: Arthur D. Little, Inc.

FIGURE 17 EXPOSURE AND ACUTE EFFECTS OF PHENOL FOR HUMANS

TABLE 41. ADVERSE EFFECTS OF PHENOL ON MAMMALS

Adverse Effect	Species	Lowest Reported Effect Level	No Apparent Effect Level
Carcinogenicity			
promotion DMBA-induced tumors	Mouse	2.5 mg/mouse 2 x week	---[a]
skin painting (alone)	Mouse	5 mg/mouse 2 x week (0.025 ml 20% soln.)	5 mg/mouse 2 x week (0.1 ml 5% soln)
oral route	Mouse Rat	--- ---	0.5% drinking water for 2 years
Chromosomal damage	Mouse	6.4 ug/kg/day (gavage)	---[a]
Teratogenicity	Rat	---	200 mg/kg (intra-peritoneally) days 9-11 or 12-14
Renal damage	Rat	50 mg/kg/day for 135 days (gavage)	50 mg/kg/day for 20 days (gavage)
Lung damage (inhalation)	Rat	---	100-200 mg/m^3 7 hrs/day, 5 days/week, for 53 days
Oral LD$_{50}$	Man	1 gram	---

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[a] Not known.

Source: Section V.

TABLE 42. HUMAN EXPOSURE TO PHENOL

Exposure Route	Exposure (mg/kg/day)	Subpopulation	Comment
Ingestion			
Drinking Water	0 0.0002	large very small	Limited to persons drinking untreated water from polluted rivers.
Food - fish and smoked meats	0.015	unknown, may be large	Assumes 16 mg/kg in fish and 29 mg/kg in smoked products; consumption of 20 mg/day of each.
fish	0.1667	very small	Assumes maximum consumption of fish of 200 mg/day; maximum of 50 mg/kg in fish.
smoked meats	0.10	very small	Assumes maximum consumption of smoked products of 200 mg/day; maximum of 29 mg/kg in smoked foods.
Phenol-containing mouthwash	1.45	may be large, maximum assumptions	Assumes use of 60 ml/day mouthwash (1.4% phenol), 10% retention, not chronic use.
Phenol-containing throat lozenges	4.33	may be large	Assumes recommended dosage - 8/day 32.5 mg total phenol/lozenge, not chronic use.
Inhalation			
Contaminated air near highways	0.10	unknown	Maximum concentration of phenol in air in Germany near highways. 0.3 mg/m^3; inhalation of 20 m^3/day.
Dermal			
Face creams	unknown; probably much less than 0.21 mg/kg	unknown, may be large	Use of 5 grams of face cream per day containing 0.5% phenol.

Source: Section VI-B.

separating exposure and effects levels is a narrow one, the significance of this apparent problem is modified by two factors. First, both the subpopulation size and exposure duration related to the largest exposure, use of medicinal lozenges, are small. Second, due to the difficulty in interpreting the study reporting the lowest effects concentration, as discussed previously, the implications of its results in terms of human risk are uncertain.

Exposure levels were estimated for several special-case subpopulations as well as for the general population (see Section V-A). All estimates assumed some exposure to contaminated water, ingestion of some contaminated food (no total diet study was available), and inhalation of upper-limit highway levels of phenol. This baseline exposure level resulted in an exposure level of 0.10 mg/kg daily for the largest fraction of the population. Any population members using skin creams containing phenol on a daily basis would increase their exposure approximately by a factor of 2 to a level of 0.22 mg/kg. Special subpopulations with higher daily exposure levels were laboratory workers (1.4 mg/kg), people consuming phenol medicinal products (5.9 mg/kg), people exposed via ingestion of contaminated groundwater (4.1 mg/kg), and a worst-case highly improbable subpopulation exposed to all major pathways for phenol (8.7 mg/kg).

As regards Figure 17, the scenario levels would cause the human ingestion curve to overlap the effects curve, although still within the region of the chromosomal damage study. The exposure levels as estimated are approximately 5-10 times less than the other adverse effects levels reported in Table 41.

The risks due to inhalation exposure are probably limited to a very small and perhaps nonexistent subpopulation. In addition, these risks are probably small since effects are observed in animals only at high concentrations relevant to ambient concentrations.

The use of phenol-containing products resulting in dermal absorption probably does not represent a risk to the consumer, except perhaps in the use of face peels, which is a medical procedure. However, phenol's role as a promoter of carcinogens suggests that the possibility of risk exists. There are numerous conditions which must be met in order for the initiation-promotion process to enhance tumor production, and the chances of these conditions being met are remote.

The one epidemiological study available concerned a phenol spill into groundwater. The incident is described in greater detail in Section IV (Pathway #5) and in Section V-B. Sublethal effects, such as diarrhea, mouth sores, and burning of the mouth, were associated with exposure levels of 0.11 to 40 mg/kg/day in drinking water (Baker et al. 1978). The exposure period was several weeks in total for most individuals. No residual abnormality was noted in exposed individuals after removal from contaminated drinking water.

The estimated exposure levels in the above scenarios fall within the range of exposure levels reported in the preceding field study. Therefore, it is assumed that sublethal effects similar to those described for this incident would result from all the scenario exposure levels calculated in Table 43. Presumably, additional effects, such as death or chronic effects, would not be caused by these levels. The only exception might be chromosomal damage such as that reported in the Bulsiewicz study (Bulsiewicz 1977). The effects associated with the epidemiology study at the reported exposure levels are consistent with the figure. Since they are sublethal effects rather than lethal (as those in the figure), they fall within the lower part of the curve, if at all.

C. AQUATIC BIOTA

1. Statement of Risk

Based on a limited set of monitoring data, it appears unlikely that phenol commonly reaches concentrations in surface water which adversely affect aquatic populations. At least an order of magnitude difference separated typical ambient surface water levels from the lowest (primarily sublethal) effects levels and there was at least a two-orders-of-magnitude difference from the lower lethal effects levels for fish. Numerous spills or accidental discharges have been documented involving phenol or phenol-containing waste. However, rapid microbial utilization of the compound appears to reduce its concentration to harmless levels in approximately one week (under acclimated environmental conditions). Therefore, the greatest risk for aquatic organisms is exposure in the vicinity of spills within a few days to a week following the incident and in any area where an active microbial population is not present.

2. Discussion

The lack of adequate monitoring data for phenol levels in surface waters precludes a detailed assessment of the national risk to aquatic populations in regard to phenol. A significant fraction of total environmental releases (30% including POTWs) are to water and, although phenol is generally rapidly degraded, there are many opportunities for short-term harm due to the widespread environmental distribution of phenol. This section discusses the potential risks of aquatic species by comparison of effects levels to monitoring data, discussion of known environmental exposure incidents and their resulting effects, and identification of industrial sources potentially contributing to situations where harm to aquatic life appears likely.

Table 44 summarizes the phenol concentration ranges described in Sections IV and VI-B to which aquatic species are likely to be exposed in the environment. Table 45 presents effects concentration ranges derived from laboratory data in Section VI-A.

TABLE 43. EXPOSURE LEVELS OF PHENOL FOR VARIOUS SUBPOPULATIONS[1]

Exposure Source	Subpopulation Size	Estimated Exposure Level[2] (mg/kg/day)
Scenario #1 (worst case)	Extremely small, very unlikely	8.7
Scenario #2 (people exposed to groundwater spill)	Very small, acute situation	4.1
Scenario #3 (users of phenol medicinal products)	Very small, acute situation	5.9
Scenario #4 (laboratory workers)	Very small	1.4
Scenario #5 (general population)	Large	0.10 0.20 with face cream

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[1] All incidents described in greater detail in Section V-A. Note that different exposure-route concentrations were combined (e.g., inhalation and ingestion) which may contribute some error to the total level.

[2] Calculated for 60-kg human.

TABLE 44. SUMMARY OF REPORTED ENVIRONMENTAL CONCENTRATIONS OF PHENOL

Medium	Concentration	Source
Ambient Surface Waters		
National average (STORET -unremarked data)	ND - 6.8 mg/l typically <0.01 mg/l	Tables 22 and 40
• mean	0.3 mg/l	Tables 22 and 40
• median	0.001 mg/l	
• 85 percentile	0.3 mg/l	
• maximum	6.8 mg/l	
Effluents		
Total range	ND - 3,016 mg/l	Table 23
Petroleum refinery[1]	0.88 -3,016 mg/l	
Spills		
River	Initially 28 mg/l[2] reduced to ∿1 mg/l[3] after 1 week	Table 17

ND = not detected.

[1] Final effluents.

[2] Of primarily monohydric phenols

[3] Typical background concentration for area

TABLE 45. SUMMARY OF EFFECTS LEVELS OF PHENOL ON AQUATIC ORGANISMS

Phenol Concentration (mg/1)	Effect
0.1 - 1.0	Subacute effects on certain freshwater invertebrates
1.0 - 10.0	Acute effects on sensitive freshwater invertebrates and both fresh and saltwater fish; subacute effects on other fish species.
10.0 - 50.0	Acute effects on all tested freshwater fish species and saltwater invertebrate[1]; acute effects on many freshwater invertebrates.
50.0 - 500	Acute effects on almost all freshwater invertebrates.

[1] Based on limited marine data

Source: Adapted from Section VI-A.

Comparison of the data from the two tables makes it evident that ambient concentrations are almost entirely below the lowest effects level of 0.1 mg/l. Some of the effluent levels exceeded the concentrations likely to affect most aquatic species; however, dilution and degradation would make the likelihood of populations encountering such high levels very small. Judging by the magnitude of the STORET observations, which may represent levels measured close to a source, the rareness of high effluent levels contributing to high surface water concentrations is apparent.

Table 46 provides information on the location of and activities associated with United States fish kills attributed either wholly or partly to phenol between 1971 and 1977. Unfortunately, no data on phenol concentrations were available in the fish kill reports; moreover, the presence of other toxic chemicals cannot be ruled out, particularly when they are not monitored. It is very likely that, in some cases, synergy between phenol and other toxicants or oxygen depletion increased the magnitude of the fish kill beyond the effect of phenol alone. Since phenol is widely used for synthesizing a variety of aromatic compounds, the chemical industries themselves and the transport of their products may continue to be the major sources of serious spills and discharges of phenol. The fish kills occurred primarily in the northeastern states in industrial areas, with a few incidents in southern and midwestern areas.

Most of the more comprehensive field studies of fish kills associated with measured concentrations of phenol are from Europe and concern phenols as a group. The proportion of the toxic effects attributable directly to phenol depends on the particular waste characteristics. However, since phenol is rarely present by itself in any waste, for an approximation of its environmental impact it is useful to examine the data available on the combined effects of phenols. It should be pointed out, though, that associated substances, such as chlorophenols, may be considerably more toxic than phenol so a combined phenol effects level can be much lower than a phenol effects level.

Table 47 presents brief descriptions of aquatic exposure incidents caused by phenols. No information was available on the sources of the wastes; however, accompanying water concentrations were measured and provide a useful set of data for comparison with U.S. surface water monitoring data.

Comparison of Table 45 and 47 provides an opportunity to confirm laboratory-derived concentrations. In the laboratory studies no effects were observed at lower than 0.1 mg/l and only subacute effects or lethal effects on sensitive species were observed between 0.1 and 10 mg/l. In the field studies, fish kills were usually reported starting at 3 mg/l (assuming that phenol comprised all the toxic waste present).

TABLE 46. DATA ON PHENOL-RELATED FISH KILLS IN U.S. (1971-1977)

Date	Water Body	Location	Number Killed	Source
5-25-71	Roaring Brook	Glastonbury, CT	-	High phenol, Zn, Cu in fish tissues No toxics measured in water
6-8-71	Casey Fork Cr.	Mt. Vernon, IL	6,000	Wood preservation
8-6-71	Tunungwant Cr.	Bradford, PA	53,000	Discharge for chemical industry in area
8-6-71	Tunugwant Cr.	NY, near Bradford, PA	45,000	From Bradford, PA
8-6-71	Allegheny R.	Irvine Mills, NY	62,000	From Bradford, PA
1971	Ohio R.	New Martinsville, WV	5,000	Phenols from nearby chemical industry
1971	Milwaukee R.	Gratton, WI	1,500	Phenols, oil from storm sewer (?)
1972	Severn Run (Branch)	Odenton, MD	100	Phenols from plastics industry
1973	Kingsland Cr.	Lyndhurst, NY	5,000	Phenolic discharge from chemical industry
5-18-74	Hardisty Pond	Southbury, CT	550	Mixed solvents, heavy oil, and phenol
5-22-74	Banmers Pond	Naugatuck, CT	010	Asphalt and phenol
6-18-74	Red Clay Cr.	Newcastle, DE	2,000	Haveg Industry phenol spill
6-19-74	New Haven Harbor	New Haven, CT	20,000	High phenol, Al, pH, BOD, and coliform
7-29-74	Black Warrior R.	Tuscaloosa, AL	10,700	17,000-21,500 lb phenol spill by Reichhold Chemical
6-17-76	Black Rock Harbor	Bridgeport, CT	25,000	Chemical, textile, metal industries, and POTW nearby; high phenol, Cu, and Zn in fish tissues
6-22-76	Bridgeport Harbor	Bridgeport, CT	20,000	Discharges from POTW, power plant
11-17-76	Great Miami R.	Ohio	0.848	Metal and cyanide production
1976	Bear Cr.	Fairview, PA	28,000	Phenols, cyanides from agric. operations
5-10-77	Hebble Cr.	Greene Co., OH	1,000	"Government operations"
6-1-77	Sanders Branch	Hampton, SC	Total	Railway phenol spill
8-2-77	Beaverdam Cr.	Damascus, VA	150	Discharge by American Cyanamid

Source: MDSD Fish Kill Survey.

TABLE 47. EXPOSURE INCIDENTS INVOLVING PHENOLICS

Concentration of Phenols (mg/1)	Effect	Reference
0.02 - 0.3	At 0.02 mg/1 in a river an abundant and diversified fish fauna was present. At 0.3 mg/1 no fish were found.	Kalabina 1935
1 - 10	In a small Luxembourg stream at 1 mg/1 fish populations including salmonids were present. When discharge increased concentrations to 10 mg/1, all fauna within a 9-km stretch were killed. Dissolved oxygen levels were 0-10% of air saturation level. Downstream at 3-10 mg/1 (D.O.10-50%) only salmonids were killed. Further downstream at 3 mg/1, no fish were killed.	Krombach and Barthel 1964
0 - 130	At lower concentrations (up to 3.2 mg/1[1]) in Yugoslavian river, fish were present. At higher levels (>3.2 mg/1) all fish were absent.	UNFAO 1973
3 - 5	Fish kills ascribed to concentrations exceeding these levels	Ludëmann 1954

[1] Actually presented as 4.4 mg/1 but the lower concentration was assumed as a conservative estimate.

However, one study (Kalabina 1935) reported no fish present at phenol concentrations of 0.3 mg/l. There was no information available on oxygen levels or other toxins present which may have influenced the order of magnitude difference from the other concentrations reported. Laboratory data (Table 45) suggest, however, that the 3-mg/l lethal threshhold (on a population scale) is more likely due to the fact that most species are killed by phenol concentrations of between 1 and 50 mg/l. Therefore these two sets of data, laboratory and field, are compatible despite all the potentially interfering factors.

A significant indirect effect of phenol discharge to aquatic systems is oxygen depletion. As discussed in Section IV, phenol is readily degradable by microorganisms which can assimilate the chemical as a sole carbon source. In addition phenol is likely to be discharged in association with other degradable organics (e.g., sulfites). The presence of phenol supports the rapid growth of microbial populations which require oxygen for oxidation processes and auxiliary activities. This can result in extreme oxygen depletion on a local scale (up to 100%) which may in turn result in the death through suffocation of invertebrates and fish. Therefore, the toxic effects of a phenol discharge may be completely or partially attributable to anaerobic conditions. Table 48 presents phenol concentrations, accompanying degrees of oxygen depletion, and the effects on an aquatic population from a field study of a phenol spill (Krombach and Barthel 1964) described in greater detail in Section IV. The greatest O_2 deficiency was associated with the highest phenol levels and was clearly responsible for some of the toxicity, especially at a ∿100% deficiency.

TABLE 48. EFFECTS OF A PHENOL SPILL ON A RIVER SYSTEM

Distance From Spill (km)	Phenol Concentration (mg/1)	Oxygen Deficiency (%)	Effect
0 - 9	>10	90 - 100	All aquatic flora and fauna present destroyed. Microbial activity on phenols and sulfites also present in waste.
9 - 19	3 - 10	50 - 90	Only salmonid population killed; slight effect on aquatic flora.
19 - 37	<3	<50	No damage.

Source: Krombach and Barthel 1964.

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APPENDIX A. BACKGROUND NOTES ON THE DERIVATION OF TABLE 2 [1]

1. U.S. International Trade Commission 1978.

2. U.S. International Trade Commission 1978 states that 93,500 kkg of phenol are produced by synthetic processes other than cumene. The other methods are listed as phenol by toluene oxidation (Kalama Chemicals, Inc.) and caustic fusion (benzene sulfonation) (Reichhold Chemicals, Tenneco Oil Co., and Sherwin Williams).

3. D. Beck. U.S. International Trade Commission, personal communication, January 1980.

4. U.S. Department of Commerce 1978a. Associated handling, storage, and transport losses are reported under the heading Producer, Storage, Loading, and Transport.

5. These data were based on the production figures reported in U.S. International Trade Commission 1978 and on the phenol end-use pattern reported by percent in Chemical and Engineering News 1978.

6. According to data supplied to U.S. EPA by Monsanto (Eimutls et al. 1978) there are a number of chemical use categories that are sources of phenolic emissions:

Source	Emission (kkg)
Residential Wood Combustion*	7,223.5
Acetone and Phenol from Cumene*	233.8
Bisphenol A*	73.9
Nonylphenol*	53.7
Pentachlorophenol and Sodium Salts*	23.6
p-Nitrophenol	22.5
Trichlorophenols*	11.3
Salicyclic Acid*	9.3
Chlorophenol*	6.4
Polyvinyl Chloride	4.5
Polycarbonate Resins	3.9
Cresyldiphenyl phosphate	3.3
Silvex	1.4
Octylphenol	0.8
Salicyclates (excluding aspirin)	0.6
TOTAL	7,673.0

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[1] Appendix based primarily on Versar 1980.

The source categories that are marked by an asterisk(*) are listed in Table 2 in the Airborne Emissions category and are marked in the table by a note #8. The sum of the emissions from the source categories not marked by an asterisk is 37 kkg, or about 0.5% of the total emissions. Since the emissions from these categories are small, and since no data were accessible on the amounts of or ways that phenol is used in these categories, they have been combined under the single heading of "Other Use Category" and the "NA" listed in the "Consumption" column refers to a small unknown amount of phenol consumed.

7. U.S. Department of Commerce 1978b.

8. These data are taken from EPA information compiled by Monsanto (Eimutls et al. 1978). Note #6 summarizes the emissions data. Numbers are rounded to the nearest integer. The Use Category of "Other Chlorophenols" is actually the sum of "chlorophenols" and "trichlorophenols" as shown in note #6; i.e., trichlorophenols (11.3 kkg) and chlorophenols (6.4 kkg) add up to 17.7 kkg, or 18 kkg. Residential wood burning is a significant source, accounting for nearly 95% of all phenol emissions listed in the Monsanto report.

9. Approximately 1,108,850 kkg of phenol were produced by cumene perodixdation (U.S. International Trade Commission 1978). Phenol airborne emissions from the column vents were estimated to be 1.474 kg/kkg of phenol produced (Hedley 1975).

   Phenol Air Emissions = (1.476 kg/kkg of phenol) (1,108,850 kkg)
   $\cong$ 1,630 kkg

10. These calculations were based on the supposition that 0.161 kg of phenol were lost per 1 kkg stored. Emissions factors were taken from Delaney and Hughes 1979.

    Consumption x 0.161 ÷ 1000 = kkg of phenol emission

    The sum of emissions factors:

    from product storage and loading (0.51) + transport (0.11) = 0.161 kg/kkg

    Fugitive emissions from pressure relief valves, pump seals, equipment purges, process drains, are not included in this estimate. No process dependent discharges have been included in the estimate.

    Thus, emissions due to the producer handling, loading, and transport are the product of the above emission factor (0.161 kg/kkg) and the total phenol production (1,216,100), or 196 kkg. (N.B. Emissions factors are averages of only two data points.)

11. The 196 kkg of emission listed in note #10 as deriving from "Producer Storage, Loading, and Transport" has been doubled as an estimate of the emissions resulting from "User Storage, Loading, and Transport".

The rationale behind this is that it is likely that since there are far more users than producers of phenol, emissions from user handling procedures probably greatly exceed the handling emissions from producers. Therefore the amount of 392 kkg emitted by user handling practices is probably conservative, if the 196 kkg emission attributed to producer handling is accurate.

12. Iron and steel production involves the use of a large amount of coal and coke, so that even though there are no data on the airborne emissions of phenol from iron and steel production (and the corresponding coal use), it is possible that the amount of phenol generated during iron and steel production is large.

13. No data are available on the phenol airborne emissions from coal-fired home furnaces, of which there are a large number used in the coal-producing parts of this country, but since this is a large source of polycyclic aromatic hydrocarbons (NAS 1972), it seems probable that phenol is also emitted, but no data could be found from which estimates of the amount of phenol produced could be made.

14. Approximately 1,108,850 kkg of phenol were produced by cumene peroxidation (U.S. International Trade Commission 1978). Phenol aquatic discharges were estimated to be 0.0755 kg/kkg of phenol produced based on raw waste levels (Hedley 1975). In a sample of 9 phenol producing plants, 4 discharge direct to surface water (3 following biological treatment), 2 to POTW's, 2 to lagoons or deep wells and 1 unknown (U.S. EPA 1980). Assuming each plant has an equal discharge (9.2 kkg phenol/plant annually), that the unknown plant discharges in treated waste to surface water and a 95% efficiency during biological treatment, then aquatic discharges can be estimated as follows.

    Phenol Discharge to Surface Water = (9.2 kkg/plant) (3 plants using biological treatment (0.05 efficiency) + (9.2 kkg/plant) (2 plants no treatment) = 20 kkg annually.

    Phenol Discharge to POTW's = (9.2 kkg/Olant) (2 plants) - 18 kkg annually.

15. Approximately 87,360 kkg of phenolic resins were produced in 1978 (U.S. International Trade Commission 1978; Versar 1980). The manufacture of phenolic resins produces significant water wastes from the following process steps: 1) water introduced with the raw material; 2) water formed as a product of the condensation reaction; 3) caustic solutions used for cleaning the reaction kettles; and 4) blow down from cooling waters (Hedley 1975). The total wastewater quantity was estimated to contain 30 kg of phenol and phenolic per ton of phenolic resin produced (Hedley 1975) and calculated as 2,620 kkg annually.

In a sample of 29 plants which produce phenolic resins, 17% pretreated and discharged their waste to POTW's, 10% pretreated and discharged to surface water, 21% discharged untreated waste to POTW's, 17% practiced 0 discharge and 35% discharged to lagoons and deep wells. Assuming a biological treatment efficiency of 95% and equal discharge for all plants, then the annual aquatic discharge can be estimated.

Phenolics discharge to Surface Water = (10%) (2,620 kkg) (0.05 efficiency) = 13 kkg annually.

Phenolics discharge to POTW's = (17%) (2,620 kkg) (0.05 efficiency) + (21%) (2,620 kkg) (0 efficiency) = 572 kkg annually.

The remaining 52% of plants are assumed to discharge 1,362 kkg of phenolic waste to lagoons, deep wells or recycle the material within the plant.

16. Approximately 105,380 kkg of bisphenol A were produced in 1978 (U.S. International Trade Commission 1978). The phenol aquatic discharge for this product process was estimated to be 7.1 kg/kkg of bisphenol A produced (Hedley 1975). The total amount discharged annually is 748 kkg. Based on the sample of phenol producers described in #14 and assuming the same discharge distribution and treatment practices for bisphenol A producers, then 33% of all plants discharge biologically treated waste to surface water 22% discharge untreated waste to surface water, 33% discharge untreated waste to POTW's and the remaining 22% discharge to deep wells or lagoons (U.S. EPA 1980). Assuming a 95% treatment efficiency, then aquatic discharges can be estimated.

Phenol Discharge to Surface Water = (33%) (748 kkg) (0.05 efficiency) + (22%) (748 kkg) = 187 kkg annually.

Phenol Discharge to POTW's = (33%) (748 kkg) = 247 kkg annually.

17. The average phenol concentration in effluents from 23 plants in the petroleum refining industry was 670 ug/l (U.S. EPA 1980). The average flow was 3,325,000 gal/day (U.S. EPA 1980). There are 284 refineries - 182 discharge to surface waters, 48 discharge to POTW's, and 54 have a zero discharge (U.S. EPA 1979a).

Phenol Discharge to Water = (670 ug/l) (3,325,000 gal/day) (3.785 l/gal) (250 days/yr) (10^{-12} kkg/ug) (182 plants) = 384 kkg

Phenol Discharge to POTW's = (670 ug/l) (3,325,000 gal/day) (3.785 l/gal) (250 day/yr) (10^{-12} kkg/ug) (48 plants) = 101 kkg

18. While no data are available on the aquatic emissions resulting from "User Storage, Loading, and Transport," it is known that 20-gallon steel drums that have contained phenol are recycled into commerce, and that each drum contains approximately a half a pound of phenol. The number of such drums that are recycled annually is unknown, and the fate of the phenol is also unknown, but it seems likely that a portion of the phenol is rinsed out with water which either runs off directly to aquatic sinks or is flushed into POTW's. The practice that is supposed to be followed by drum recyclers is neutralization of the phenol with lye and then collection and landfill disposal of the resultant solution of sodium phenolate, but there is evidence that this practice is not universally followed (J. Warring, James T. Warring and Sons Barrel Company, personal communication, November 1979).

19. Phenol was detected in the effluent of the timber products processing industry during the verification sampling and analysis program (U.S. EPA 1980). Phenol is discharged in significant amounts from three subcategories - wood steaming, hardboard S2S, and insulation-thermochemical pulping and refining:

Subcategory	# of Plants Direct	# of Plants POTW's	Avg. Flow (gal/day)	Conc. (ug/l)	Discharge (kkg)
Wood Steaming	--	13	9,600	15,900	2
S2S	3	--	4,548,170	100	1
Insulation-- Thermochemical Pulping & Refining	4	--	13,803,190	17	1
TOTAL					4

20. Phenol was detected in the effluents of the leather tanning industry during the verification sampling and analysis program (U.S. EPA 1980). Phenol is discharged in significant amounts from three subcategories:

Direct Dischargers (U.S. EPA 1980 ; U.S. EPA 1978)

Subcategory	# of Dischargers	Avg. Flow (gal/day)	Avg. Conc. (ug/l)	Discharge (kkg)
Hair Pulp, Chrome Tan Retan-Wet Finish	3	600,000	1,500	3
Hair Save, Chrome Tan Retan	3	300,000	920	1
No Beamhouse	3	120.000	6,200	2
			TOTAL	6

(Data are an average of one to four plants per subcategory)

Dischargers to POTW's (U.S. EPA 1980 ; U.S. EPA 1978)

Subcategory	# of Dischargers	Avg. Flow (gal/day)	Avg. Conc. (ug/l)	Discharge (kkg)
Hair Pulp, Chrome Tan Retan Wet-Finish	71	475,000	1,500	48
Hair Save, Chrome Tan Retan	21	300,000	920	5
No Beamhouse	24	75,000	6,200	11
			TOTAL	64

(Data are an average of one to four plants per subcategory)

21. Phenol was detected in significant quantities in two subcategories of the textiles industry -- woven fabric finishing simple, and woven fabric finishing (U.S. EPA 1980).

Subcategory	Avg. Conc. (ug/l)	Total Flow Dis-charges* (MGD)	Phenol Discharge to Water (kkg)	Total Flow for Indirect Discharges (MGD)	Phenol Discharge to POTW's (kkg)
Woven Fabric Finishing Simple	15	15.5	0.2	36.5	0.5
Woven Fabric Finishing	9	58.3	0.5	40	0.3

Total Discharge to Water = 0.2 kkg + 0.5 kkg = 0.7 kkg ≈ 1 kkg

Total Discharge to POTW's = 0.5 kkg to 0.3 kkg = 0.8 kkg ≈ 1 kkg

*U.S. EPA 1979b.

22. Data were available for 9 of the 15 subcategories in the iron and steel industry. Phenol was detected in significant quantities from two subcategories - byproduct coking and cold rolling.

For the byproduct coking subcategory there are 32 direct dischargers, 21 indirect dischargers, and 17 zero dischargers (U.S. EPA 1979c). The total water usage for the subcategory was 71.1 MGD (U.S. EPA 1979d). The average phenol concentration from 3 plants was 0.026 mg/l.

Phenol Aquatic Discharge
Byproduct coking = (0.026 mg/l) (71.1 x 10^6 gal/day) (3.785 l/gal) (250 days/yr) (10^{-9} kkg/mg)
= 1.8 kkg

Direct Discharge = (1.8 kkg) $\frac{32 \text{ plants}}{32 + 21 \text{ plants}}$
= 1 kkg

Discharge to POTW = 1.8 kkg - 1 kkg = 0.8 ≈ 1 kkg

For the cold rolling subcategory, the total flow for the subcategory is 252.5 MGD. There are 230 direct dischargers, 24 indirect dischargers, and 15 zero discharges (U.S. EPA 1979d). The average concentration from three plants is 0.008 mg/l (U.S. EPA 1979e).

Phenol Aquatic Discharge From Cold Rolling
= (0.008 mg/l)(252.5 x 10^{-6} gal/day)(3.785 l/gal)(250 days/yr) (10^{-9} kkg/mg)
= 1.9 kkg

Direct Discharge = (1.9 kkg) $\frac{230 \text{ plants}}{230 + 24 \text{ plants}}$ = 1.7 kkg ≈ 2 kkg

Discharge to POTW = 1.9 kkg - 1.7 kkg = 0.2 kkg ≈ negligible

Thus the total direct discharge for the iron and steel industry = 3.0 kkg while the discharge to POTW's = 1.0 kkg.

23. The average phenol concentration in effluents from 13 plants in the ash handling subcategory of the steam electric industry was 4 ug/l (U.S. EPA 198c). There are 777 plants that discharge ash handling water to surface waters and the average flow is 2.24 x 10^6 gal/day (U.S. EPA 1980c).

Phenol Discharge to Water = (4 ug/l)(2.24 x 10^6 gal/day)(777 plants) (365 day/yr)(3.785 l/gal)(10^{-12} kkg/ug)
= 10 kkg

24. A survey of discharge monitoring reports from 10 sewage treatment plants indicated that phenol was present in the effluents at a concentration of 0.128 mg/l (Versar 1978). The total daily flow from all POTW's within the United States is estimated to approximate 22,670 MGD.

Phenol Discharged to Water = (0.128 mg/l)(22,670 x 10^6 gal/day) (365 days/yr)(3.785 l/gal) (10^{-9} kkg/mg = 4,000 kkg/yr

25. In these production and use categories there is only one plant. All the effluent waters are directly discharged after treatment by a wastewater treatment train. None of the waters are discharged to POTW's (Maria Irizarry, U.S. Environmental Protection Agency, personal communication, January 1980).

26. Approximately 1,108,850 kkg of phenol were produced by cumene peroxidation (U.S. International Trade Commission 1978). Phenol solid wastes from the evaporator residue were estimated to be 0.75 kg/kkg of phenol produced (Hedley 1975).
Phenol Solid Wastes Discharge = (0.75 kg/kkg of phenol)(1,108,850 kkg)
= 832 kkg

27. Approximately 93,500 kkg of phenol are produced by benzene sulfonation and toluene oxidation (U.S. International Trade Commission 1978). According to the SRI Directory of Chemical Producers, the maximum capacity for benzene sulfonation is 70,293 kkg (SRI 1978). Phenol solid wastes from this production method were estimated to be of phenol produced (Hedley 1975).

Phenol Solid Waste Discharge = (3.7 kg/kkg of phenol)(107,250 kkg)
= 397

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APPENDIX B: PHENOL AND PHENOLIC RESIN PRODUCTION

This appendix describes in detail the production processes for synthesizing or extracting phenol and the process for producing phenolic resins. The processes for synthesizing or extracting phenol described are: 1) recovery from coal tar and petroleum streams, 2) cumene peroxidation, 3) benzene sulfonation, and 4) toluene oxidation. This information can be used to supplement that contained in Section III of this report (Materials Balance) and to help explicate the potential losses of phenol to the environment during production.

RECOVERY OF PHENOL FROM COAL TAR AND PETROLEUM STREAMS

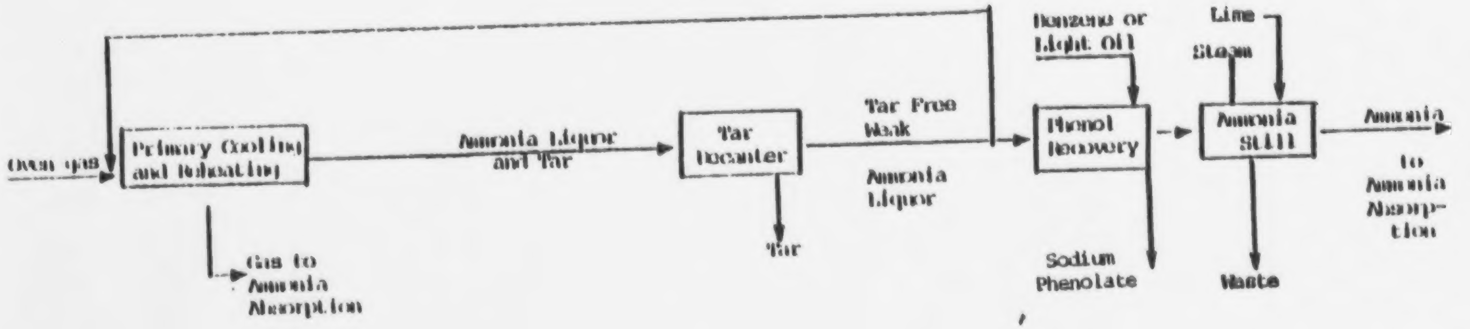
About 12,200 kkg (1%) of the phenol consumed in the United States is natural phenol derived from coke-oven operations, coal-tar distillation, and petroleum operations.

Phenol From Coal Tar

Although most coke-oven plants in the United States are equipped to process tar and light oil, the extent to which an individual plant produces the various products depends on the size of the plant and its economic condition (U.S. EPA 1977). Figure B-1 presents a segment of the coke byproduct recovery plant. The processes shown are primary cooling/reheating, tar decanting, phenol recovery, and the ammonia still.

In the coke-oven process, the hot gases resulting from coke carbonization are collected and sprayed with weak ammonia liquor to reduce the temperature and volume. The tar is condensed and decanted, along with the unevaporated liquor, into a decanter. The coke-oven gas and uncondensed vapors are further cooled in a primary cooler. As the gas cools, tar and ammonia liquor are condensed. This mixture of condensed liquor flows to the decanter. The gas, with a nominal amount of tar fog, is sent to an electrostatic precipitator through a steam-driven centrifugal gas exhauster. The precipitator removes the final traces of tar, and the gas is reheated and passed through an ammonia scrubber. The collected tar is added to the decanter.

In the decanter, the tar is separated from the weak ammonia liquor. Part of this liquor is pumped back to the gas-collecting main sprays, another part is passed to the cooling coils of the primary cooler, and the remainder is sent to the ammonia still. The pitch sludges settle to the bottom of the decanter and are mechanically raked out for disposal, usually by burning as fuel. Settled crude tar, which contains a large number of chemical compounds, is sent to a separate plant for secondary processing by distillation, from which naphthalene is obtained as the main product.


Oven gas
Primary Cooling and Reheating
Gas to Ammonia Absorption
Ammonia Liquor and Tar
Tar Decanter
Tar
Tar Free Weak
Ammonia Liquor
Benzene or Light Oil
Phenol Recovery
Sodium Phenolate
Lime
Steam
Ammonia Still
Waste
Ammonia
to Ammonia Absorp-tion

Source: U.S. EPA 1977.

FIGURE B–1 SEGMENT OF COKE BYPRODUCT RECOVERY PLANT

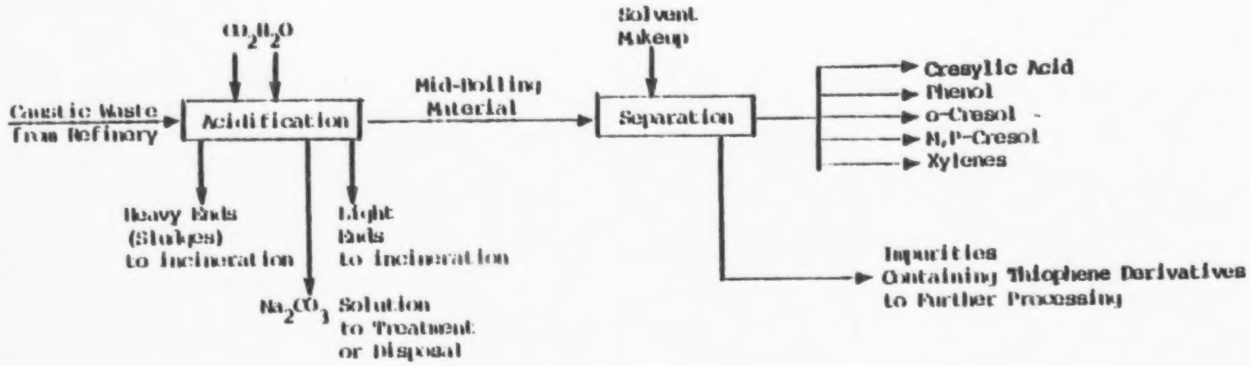
Two processes are available to recover phenols: the solvent extraction process and the vapor recirculation process (U.S. EPA 1977). In the solvent extraction process, weak ammonia liquor recovered with the volatile products of coal carbonization is contacted countercurrently in a scrubber with benzene or light oil to remove phenol. The weak ammonia liquor and light oil flow are maintained in the ratio of approximately 1.25 oil to 1.0 liquor. The phenol-free liquor flows to a storage tank for further processing. The phenolized benzene or light oil is washed with caustic soda in a tower. After a week or two, the caustic in the light-oil caustic washer is saturated with sodium phenolate, which is drained into a carbolate concentrator. The sodium carbolate in the concentrator is boiled to remove entrained solvent and moisture. It is then neutralized with carbon dioxide to liberate crude phenols and phenol compounds (U.S. EPA 1977).

The vapor recirculation method is operated in conjunction with the ammonia still. This method is only practiced if the weak liquor is not dephenolized by solvent extraction before being fed to the ammonia still. The ammonia present in weak liquor is in two forms, classified as "free" and "fixed." The free ammonia is that which dissociates readily by heat, such as ammonia carbonates, sulfides, and cyanides. The fixed ammonia requires the presence of strong alkali to effect displacement of the ammonia from the compound in which it is present; examples include ammonia chloride and sulfate (U.S. EPA 1977). In the ammonia still, free ammonia and acidic gases are removed by passing weak ammonia liquor down through a column over a series of plates equipped with bubble caps and overflow pipes. The liquor is heated by steam which vaporizes ammonia and volatile acidic gases. Ammonia is removed from the weak liquor in the "free-leg" ammonia still. The liquor leaving the base of the free-leg is transferred to the dephenolizing unit where phenols are removed by vaporization with steam followed by extraction with caustic soda. Phenols are recovered from the sodium phenolate solution. The dephenolized liquor is transferred to the "fixed leg" of the ammonia still (U.S. EPA 1977).

Phenol may enter the environment from this process in particulate matter emitted through leaks in the equipment and from the still, and in the wastewater generated by the scrubbers and the washers. The waterborne wastes from this process are usually treated before they are discharged.

PHENOL FROM PETROLEUM-REFINERY CAUSTIC WASTES

Caustic waste from refineries is the feed stream to this process. This feed stream may contain over 20% phenol along with other tar acids and thiols. The recovery of phenols from this stream is carried out in two major operations: acidification and separation. Each operation encompasses several processing steps. A general flow diagram of both is shown in Figure B-2.


CO_2, H_2O
Caustic Waste from Refinery
Acidification
Heavy Ends (Sludges) to Incineration
Light Ends to Incineration
Na_2CO_3 Solution to Treatment or Disposal
Mid-Boiling Material
Solvent Makeup
Separation
Cresylic Acid
Phenol
o-Cresol
M,P-Cresol
Xylenes
Impurities Containing Thiophene Derivatives to Further Processing

Source: Radian 1977.

FIGURE B–2 RECOVERY OF PHENOL FROM PETROLEUM REFINING CAUSTIC WASTES

There may be three processing steps involved in acidification: preparation, springing, and separation.

In the preparation process, the feed stream may be devolatilized to remove the remaining hydrocarbon gases which may be vented or flared. In some plants, the mercaptans and thiols are oxidized with air and steam under alkaline conditions to convert the sulfur compounds to disulfides. The effluents of this step are sent into a settling tank where the separation is made by decanting. The disulfide layer is incinerated. Other plants do not have a preparation step, and the raw feed is sent to springing instead.

The prepared or raw feed is sent to a packed springing tower in which the acidification takes place. The feed stream is contacted countercurrently with CO_2 in the form of flue gas. Resulting cresylates and sodium phenates are converted to cresols and phenols.

The acids are decanted from the water in settling tanks. The phenolic layer enters a fractionation column in which light and heavy ends are removed and incinerated. The mid-boiling range material is sent to the solvent extraction unit. The aqueous sodium carbonate layer contains phenols whose concentrations are reduced by absorption and stripping. The resultant wastewaters contain phenols and require careful disposal and treatment (Hawley 1977).

Solvent extraction is typically done with aqueous methanol and naptha. The phenols are concentrated in the methanol layer, while the naptha removes most of the impurities. The methanol stream is stripped before the phenolic stream is pumped to a separating tank where water is removed. The acid stream is then fed to a series of fractional distillation columns for the separation of phenol, cresols, and xylenols. An ion-exchange column may be used to remove traces of mercaptans and bases from the phenolic compounds (Hawley 1977).

The aqueous methanol extraction step generates wastewater contaminated with phenolic compounds. There is no available information on the disposal or treatment of this wastewater.

PHENOL FROM CUMENE PEROXIDATION

The manufacture of phenol by the cumene peroxidation process involves the liquid-phase air oxidation of cumene to cumene hydroperoxide, which is then decomposed to phenol and acetone by the addition of acid (Lowenheim and Moran 1975)

The basic reaction steps are:

1. $\underset{\text{cumene}}{C_6H_5CH(CH_3)_2} + 2O_2 \longrightarrow \underset{\text{cumene hydroperoxide}}{C_6H_5C(CH_3)_2OOH}$
2. $C_6H_5C(CH_3)_2OOH \xrightarrow{\text{acid}} \underset{\text{phenol}}{C_6H_5OH} + \underset{\text{acetone}}{CH_3COCH_3}$

Cumene (isopropylbenzene) is produced by liquid- or vapor-phase alkylation of benzene with propylene.

To produce phenol, cumene is mixed with recycled cumene, purified, and then charged to the oxidation reactor along with dilute soda ash to maintain the pH between 6.0 and 8.0. The mixture is oxidized with air at 110 to 115°C until 20 to 25% of the cumene is converted to the hydroperoxide intermediate. The cumene/hydroperoxide mixture is concentrated in an evaporator and then fed to a reactor in which the cumene/hydroperoxide is cleaved to phenol and acetone in the presence of a small amount of sulfuric acid. The typical operating temperature of this reaction is 70 to 80°C, and the resulting mixture consists of acetone, acetophenone, α-methylstyrene, and cumene.

The products are separated by distillation. In the process shown in Figure B-3, acetone is removed in the first column and further purified. The bottoms from the first column are vacuum distilled, and unreacted cumene and α-methylstyrene are taken overhead. This stream is further purified by catalytic hydrogenation to convert the α-methylstyrene to cumene. In some plants, α-methylstyrene is carefully fractionated and is available as a byproduct.

The bottoms from the vacuum still are further fractionated to separate phenol and acetophenone, yielding about 90% phenol (Lowenheim and Moran 1975).

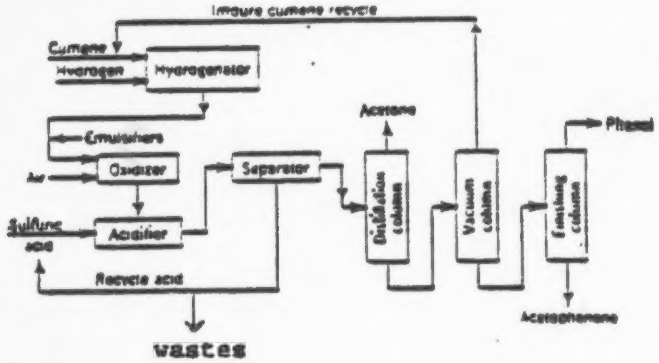
Estimated total environmental emissions of phenol from this process are on the order of 2,000 kkg. Airborne emissions are from the sulfuric acid column vents. Contaminated wastewater comes from the phenol surge vessel. Solid wastes contaminated with phenol are from the residues in the evaporation vessels (Hedley 1975).

PHENOL FROM BENZENE SULFONATION

This process involves the reaction of benzene with sulfuric acid followed by the conversion of the benzene sulfonic acid to phenol.

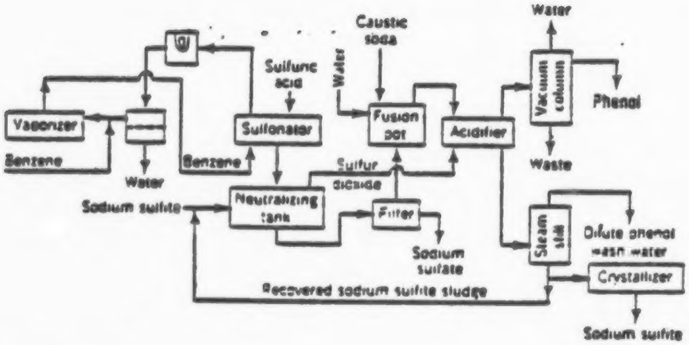
The benzene sulfonic acid is prepared by reacting concentrated sulfuric acid with benzene. Water is formed during the reaction and must be removed to prevent its diluting the sulfuric acid. When the concentration of sulfuric acid drops below 78%, the sulfonating reaction stops. This reaction is carried out continuously in vapor phase by passing benzene vapors up through the reaction zone maintained at 150°C. The sulfonation proceeds until only a few percent of the sulfuric acid remains. It is then neutralized. The benzene, water, and acid vapors are condensed and the benzene recovered.

The sulfonation product is added rapidly to a neutralizing tank which contains sodium sulfite. The sulfur dioxide formed is boiled off and piped to the acidifiers. The boiling mixture of sodium benzene sulfonate and sodium sulfate is filtered. The sodium sulfate precipitates


Impure cumene recycle
Cumene
Hydrogen
Hydrogenator
Emulsifiers
Air
Oxidizer
Sulfuric acid
Acidifier
Separator
Recycle acid
Acetone
Distillation column
Vacuum column
Finishing column
Phenol
Acetophenone
wastes

Source: Lowenheim and Moran 1975.

FIGURE B–3 CUMENE PEROXIDE PROCESS


Water
Caustic soda
Sulfuric acid
Water
Vaporizer
Benzene
Water
Benzene
Sulfonator
Fusion pot
Acidifier
Vacuum column
Phenol
Waste
Sulfur dioxide
Sodium sulfite
Neutralizing tank
Filter
Sodium sulfate
Steam still
Dilute phenol wash water
Crystallizer
Recovered sodium sulfite sludge
Sodium sulfite

Source: Lowenheim and Moran 1975.

FIGURE B–4 BENZENE SULFONATE PROCESS

out of the hot liquor and remains on the filter. The sodium benzene sulfonate is pumped into a fusion pot that contains fused caustic soda to yield sodium phenate. A ratio of 3 moles of alkali to 1 mole of sulfonate is used. After the fusion is complete, the pot is emptied, and the melt is diluted with water. The sodium phenate-sodium hydroxide-sodium sulfate solution is acidified with sulfur dioxide and a small amount of sulfuric acid. The crude phenol separates as the upper layer over the aqueous solution of sodium sulfite and sodium sulfate. The phenolic layer is decanted and distilled under vacuum to produce refined phenol. The aqueous layer is treated with steam to remove residual phenol. The distillate is used as makeup water. Part of the sulfide sludge is used for the neutralization step, and the remainder is crystallized and dried to yield sodium sulfite byproduct. Phenol of UPS quality is obtained in about 85% by weight yield based on benzene.

The reaction steps for phenol production by benzene sulfonation are given below. A simplified flow diagram of the process is shown in Figure B-4.

$$C_6H_6 + H_2SO_4 \longrightarrow C_6H_5SO_3H + H_2O$$

$$2C_6H_5SO_3H + Na_2SO_3 \longrightarrow 2C_6H_5SO_3Na + SO_2 + H_2O \; (+ Na_2SO_4)$$

$$C_6H_5SO_3Na + 2NaCH \longrightarrow C_6H_5ONa + Na_2SO_3 + H_2O$$

$$2C_6H_5ONa + SO_2 + H_2O \xrightarrow{H_2SO_4} 2C_6H_5OH + Na_2SO_3 \; (+ Na_2SO_4)$$

Presently only one company uses this method to produce phenol, and the only identifiable waste stream generated by this process is the bottoms of the phenol columns containing phenols which is discharged as solid waste (Hedley 1975).

PHENOL FROM TOLUENE OXIDATION

Less than 2% of the phenol consumed is produced by toluene oxidation. Toluene is converted to phenol in two successive liquid-phase, air-oxidation steps, as shown in Figure B-5 (Lowenheim and Moran 1975).

In the first step, toluene in the presence of cobalt naphthenate catalyst is converted to benzoic acid. The reaction conditions are maintained at 150° to 250°C and at a pressure of 5 to 50 atms (Lowenheim and Moran 1975).

In the second step, the benzoic acid is melted in biphenyl, mixed with a small amount of manganese-promoted cupric benzoate, and fed to a reactor. Air and steam are fed into the reactor where the benzoic acid is oxidized to phenol. The reactor is maintained at 230°C and 20 to 25 psi. Phenol and benzoic acid are vaporized continuously and passed to the primary distillation column. Conversion of benzoic acid in the oxidizer is 70 to 80%; phenol yield is 90% (Lowenheim and Moran 1975).

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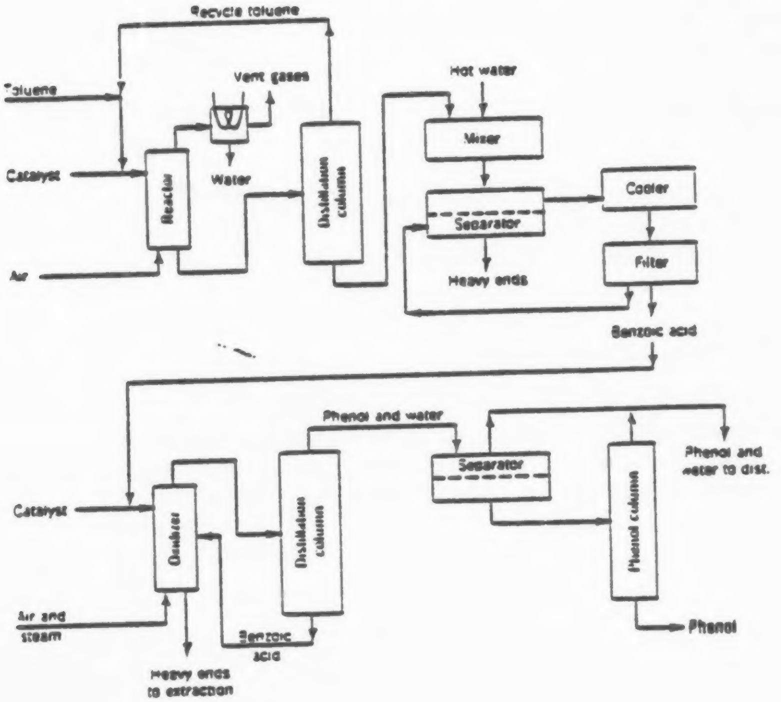

Recycle toluene
Toluene
Vent gases
Catalyst
Reactor
Water
Distillation column
Air
Hot water
Mixer
Separator
Heavy ends
Cooler
Filter
Benzoic acid
Phenol and water
Separator
Phenol and water to dist.
Catalyst
Oxidizer
Distillation column
Phenol column
Air and steam
Benzoic acid
Heavy ends to extraction
Phenol

Source: Lowenheim and Moran 1975.

FIGURE B–5 TOLUENE OXIDATION PROCESS

In the primary distillation column, phenol and water vapor pass overhead and the benzoic acid is removed from the bottom of the column and returned to the oxidizer. The condensed phenol-water mixture from the primary column separates into two layers. The lower, phenol-rich layer is sent to the phenol column where product phenol leaves the bottom of the column and water is removed from this layer azeotropically. Phenol is removed from the water layer from the azeotrope by further fractionation. The main reactions of this process are given below.

$$2C_6H_5CH_3 + {}^3O_2 \xrightarrow[\text{catalyst}]{\text{cobalt}} \underset{\text{benzoic acid}}{2C_6H_5OOOH + 2H_2O}$$

$$2C_6H_5COOH + O_2 \xrightarrow[\text{catalyst}]{\text{copper}} \underset{\text{phenol}}{2C_6H_5OH + 2CO_2}$$

Only one plant presently uses the toluene oxidation process. The economy of it depends on how cheaply toluene can be converted to benzene (Hedley 1975). Information on discharges from this plant is not available.

PHENOLIC RESIN PRODUCTION PROCESS

Table B-1 lists the manufacturers of phenolic resins and their locations. There are two major types of phenolic resins produced: resol and novalaks (Sittig 1975). Resols are a mixture of phenol and formaldehyde with an excess of formaldehyde. The mole ratio is usually 1.5 moles of formaldehyde to 1 mole of phenol. Sodium hydroxide is normally used to catalyze the polymerization reaction which takes place at a pH of 8 to 11 (Sittig 1975).

The reacting mixture contains sufficient formaldehyde to form a cross-linked thermoset resin. The reaction, however, is stopped short of completion at an average molecular weight of the polymer appropriate for the end use of materials (Sittig 1975).

Three classes of products are produced under the general headings of resols. The first is a water-soluble bonding resin which is either sold as is or neutralized and partially dehydrated. The second is a water-insoluble resin which is vacuum dehydrated and dissolved in solvents to produce laminating resins and varnishes. The third class of product is similar to the second class but the water is removed and the reaction is carried further to produce a "one-stage" solid resin which is then vacuum dehydrated and removed from the reactor for cooling and solidifying. This type of resin is either made into bonding compounds and surface coatings by adding catalysts and lubricants, or converted into thermosetting molding powders by adding catalysts, lubricants, pigments, and fillers. The compounding may be performed in the same facilities or may be shipped to custom compounders (Sittig 1975).

TABLE B-1. U.S. MANUFACTURERS OF PHENOLIC RESINS

Company	Location
Allied Products Corp Acme Chems. Div.	New Haven, CT
American Cyanamid Co. Formica Corp., subsid.	Evandale, OH
American Hoechst Corp. Indust. Chems. Div.	Mount Holly, NC
Ashland Oil, Inc. Ashland Chem. Co., Div. Foundry Products Div. Resins and Plastics Div.	Hammond, IN Cleveland, OH Calumet City, IL Fords, NJ Pensacola, FL
The Bendix Corp. Friction Materials Div.	Troy, NY
Borden, Inc. Borden Chem. Div. Adhesives and Chems. Div. - East	Bainbridge, NY Demopolis, AL Diboll, TX Fayetteville, NC Sheboygan, WI
Adhesives and Chems. Div. - West	Fremont, CA Kent, WA LaGrande, OR Missoula, MT Springfield, OR
Brand-S Corp. Cascade Resins, Inc., Div.	Eugene, OR
California Resins and Chem. Co., Inc.	Vallejo, CA
The Carborundum Co. Polymers Venture	Niagara Falls, NY
Carrier Corp. Inmont Corp. subsid.	Anaheim, CA Cincinnati, OH Detroit, MI
Chagrin Valley Co. Ltd. Nev mar Corp., subsid.	Odenion, MD

TABLE B-1. U.S. MANUFACTURERS OF PHENOLIC RESINS
(continued)

Company	Location
Champion International Corp. U.S. Plywood Div.	Anderson, CA
Clark Oil & Refining Corp. Clark Chem. Corp., subsid.	Blue Island, IL
Hercules, Inc. Haveg Indust., Inc., subsid. Marshallton Operation	Wilmington, DE
Heresite & Chem. Co.	Manitowoc, WI
Inland Steel Co. Inland Steel Container Co., Div.	Alsip, IL
International Minerals & Chem. Corp. Aristo International Corp., subsid. Foundry Products Div.	Detroit, MI
The Ironsides Co.	Columbus, OH
Knoedler, Alphonse & Co. Knoedler Chem. Co., subsid.	Lancaster, PA
Koppers Co., Inc. Organic Materials Div.	Petrolia, PA
Kordell Indust.	Mishawaka, IN
Lawter Chems., Inc.	South Kearny, NJ
Libbey-Owens-Ford Co. LOF Plastic Products, subsid.	Auburn, ME
Masonite Corp. Alpine Div.	Gulfport, MS
Monagram Indust., Inc. Spaulding Fibre Co., subsid.	De Kalb, IL Tonawanda, NY
Monsanto Co. Monsanto Chem. Intermediates Co. Monsanto Plastics & Resins Co.	Addyston, OH Chocolate Bayou, TX Eugene, OR Santa Clara, CA Springfield, MA

TABLE B-1. U.S. MANUFACTURERS OF PHENOLIC RESINS (continued)

Company	Location
Napko Corp.	Houston, TX
Occidental Petroleum Corp. Hooker Chem. Corp., subsid Hooker Chems. and Plastics Corp., subsid. Durez Div.	Kenton, OH North Tonawanda, NY
Onyx Oils & Resins, Inc.	Newark, NJ
Owens-Corning Fiberglass Corp. Resins and Coatings Div.	Barrington, NJ Kansas City, KS Newark, OH Waxahachie, TX
Plastics Engineering Co.	Sheboygan, WI
Polymer Applications, Inc.	Tonawanda, NY
Polyrez Co., Inc.	Woodbury, NJ
Raybestos-Manhattan, Inc. Adhesives Dept	Stratford, CT
Rechhold Chems., Inc.	Andover, MA Cartaret, NJ Detroit, MI Houston, TX Kansas City, KS Moncure, NC South San Francisco, CA Tacoma, WA Tuscaloosa, AL White City, OR
Varcum Chem. Div.	Niagara Falls, NY
Rogers Corp.	Manchester, CT
Schenectady Chems., Inc.	Oyster Creek, TX Rotterdam Junction, NY Schenectady, NY
Simpson Timber Co. Chems. Div.	Portland, OR

TABLE B-1. U.S. MANUFACTURERS OF PHENOLIC RESINS (continued)

Company	Location
Synres Chem. Corp. Shanco Plastics & Chems., subsid.	Tonawanda , NY
Synthane-Taylor Corp.	Betzwood, PA
Union Carbide Corp.	Bound Brook, NJ Elk Grove, CA Marietta, OH Texas City, TX
United-Erie, Inc.	Erie, PA
Univar Corp. Pacific Resins & Chems., Inc., subsid.	Eugene, OR Newark, OH Portland, OR Richmond, CA
Valentine Sugars, Inc. Valite Div.	Lockport, LA
West Coast Adhesives Co.	Portland, OR
Westinghouse Electric Corp. Insulating Materials Div.	West Mifflin, PA Longview, WA
Weyerhaeuser Co.	Marshall, WI

Source: Versar 1980.

The resols are only partially polymerized when they are manufactured. These materials, however, contain sufficient formaldehyde to carry the reaction to completion. The polymerization is completed when the resin is heated and set into the final product at the consumer facility (Sittig 1975).

Novalaks are the second category of phenolic resins. These are formed by reacting a mixture which contains a deficiency of formaldehyde. The mole ratio is 0.75 to 0.90 moles of formaldehyde to 1.0 mole of phenol. Polymerization is carried out in an acid medium using a catasyst such as sulfuric acid. The pH of the reaction ranges from 0.5 to 1.5. Since the reacting mixture contains a deficiency of formaldehyde, essentially all of the formaldehyde is consumed during polymerization. Since no further polymerization can take place, the product is a low-molecular weight, thermoplastic, stable material. The water which enters with the formaldehyde and the water of reaction are removed under vacuum, producing a solid, meltable material (Sittig 1975).

In order to complete the polymerization, the user must add additional formaldehyde or hexamethylenetetramine. With the latter material, ammonia is evolved from the reacting mass, leaving the same types of methylene linkages as can be obtained by using additional formaldehyde (Sittig 1975).

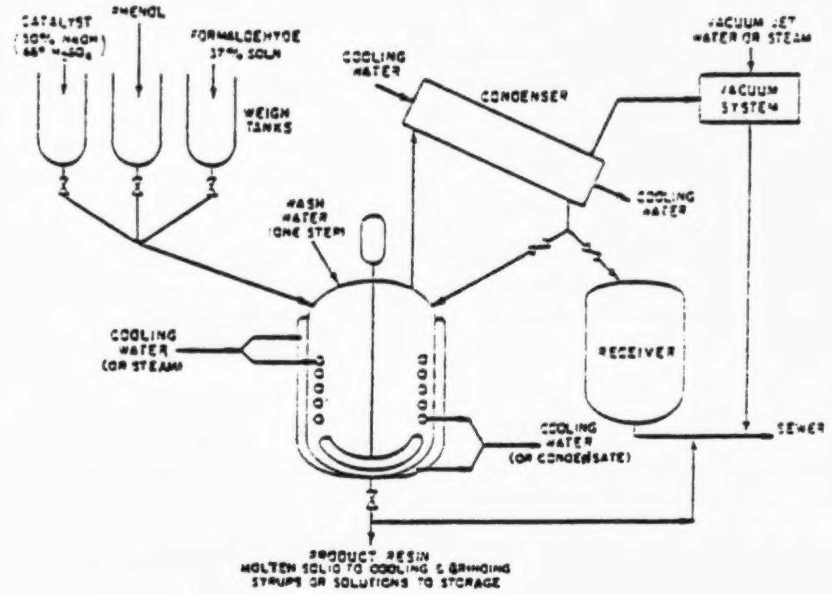
The basic resins are sometimes modified by the use of materials such as drying oils or epoxy compounds in the final stages of polymerization.

Phenolic resins are produced by batch operations because the plastic industry calls for a wide variety of polymers. There is rarely a big enough demand for a single grade of polymer to justify using a continuous process (Sittig 1975).

A schematic diagram for phenolic resin production is shown in Figure B-6. The reaction is usually carried out in a jacketed kettle. In the larger-size kettles, internal cooling coils are used to provide an adequate surface-to-volume ratio for the removal of the heat generated during the polymerization. These kettles are agitated and can operate under pressure or vacuum conditions.

The feed system consists of two weigh tanks, one each for phenol and formaldehyde solution. Commercial formaldehyde solution at 37% by weight formaldehyde is usually employed. This solution often contains about 5% methanol, which acts as a stabilizer. The kettle is equipped with a water-cooled condenser, which is also joined to a vacuum system.

For resol resin production, the phenol is charged in a molten form to the kettle, followed by the addition of formaldehyde which washes the residual phenol out of the lines leading to the kettle. A sodium hydroxide catalyst solution is next added and the kettle is heated by


CATALYST
(30% NaOH)
(66° H_2SO_4)
PHENOL
FORMALDEHYDE
37% SOLN
WEIGH TANKS
COOLING WATER
CONDENSER
VACUUM JET
WATER OR STEAM
VACUUM SYSTEM
COOLING WATER
WASH WATER
(ONE STEP)
COOLING WATER
(OR STEAM)
RECEIVER
COOLING WATER
(OR CONDENSATE)
SEWER
PRODUCT RESIN
MOLTEN SOLID TO COOLING & GRINDING
SYRUPS OR SOLUTIONS TO STORAGE

Source: Lowenheim and Moran 1975.

FIGURE B–6 PHENOLIC RESIN PRODUCTION

steam to bring the mixture to the reaction temperature of 60°C. When the condensation reaction starts, the reaction becomes highly exothermic. Thus, the supply of steam is stopped and the coils are supplied with cooling water. The mixture is held at 60°C for about 3 to 5 hours. During this period the temperature is controlled by supplying cooling water through the coils and by using total reflux returning from the water-cooled condenser on the kettle. When the polymerization reaches the desired degree, the mixture is cooled to about 35°C and the caustic solution is neutralized by sulfuric acid to a pH of 7.

The mixture is then heated again by steam to purify the resin by distillation. The water from this distillation is a concentrated waste which contains phenol, formaldehyde, and low molecular-weight resin and is usually segregated for disposal by incineration. The batch is then dumped. A few resins, such as varnish-type resols, are washed two or three times, thus generating a considerable amount of wastewater. If a resin is required to contain a very small quantity of water, a vacuum is usually applied during the latter part of the dehydration cycle. This technique is also used to produce anhydrous melt of a single-step resin.

The manufacture of novolak resins is similar to that of resols except that an acid catalyst is added at the start of the batch and a vacuum reflux is used to maintain temperatures at 85° to 90°C.

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